Appendix J

Human Health and Ecological Risk Assessment

Toxicological Risks to Ecological and Human Receptors from the
Proposed Use of Rotenone to
Eradicate Northern Pike in Lake Davis, California

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J.1 SUMMARY

This screening level risk assessment examines the ecological and human health risks for the general public that are potentially associated with the proposed use of rotenone to eradicate northern pike (*Esox luscius)* in Lake Davis, California, as defined in Section 2 of the EIR/EIS. The appropriateness of the use of rotenone for fisheries management was evaluated in a previously published Programmatic Environmental Impact Report [PEIR] (DFG 1994). The use of rotenone to achieve fisheries management goals is supported by sections 1700 and 5501 of the California Fish and Game Code, as upheld by California's Fifth District Court of Appeals (Churchill v. Parnell 1985). The continued use of rotenone as a piscicide was affirmed in the re-registration of the chemical for this use by the USEPA (USEPA 1995). Notwithstanding this background, CEQA guidance and DFG policy recognizes that site-specific analyses are still required to examine the potential effects of rotenone applications deemed to have a potential effect on non-target, non-aquatic species and special status species, as effects to these species were not fully assessed in the PEIR prepared for the general use of rotenone for fisheries management (DFG 1994).

Because the ecological and human health assessments are integrated in this appendix, general guidance from both standard ecological risk assessment protocols (USEPA 1998a; ASTM 1997; CEPA 1996) and human health protocols (USEPA 1991a,b,c; USEPA 1998b) will be evident. Thus, a standard outline of the assessment is observed that includes four key sections: problem formulation, hazard assessment, exposure assessment, and risk characterization. Elements of uncertainty in all conclusions are condensed within the risk characterization chapter.

This screening level assessment examines only the potential *toxicological* impacts from the use of the chemical formulations to ecological receptors and human populations from the rotenone formulations proposed for use in Lake Davis, as outlined in the Proposed Project and four other treatment alternatives of the EIR/EIS. Other impact analyses are provided in the body of the EIR/EIS (e.g., noise, traffic, economic, etc.), consistent with CEQA guidance, Pub. Res. Code sec. 21083. Findings in this appendix are integrated into the EIR/EIS, in Section 14 and in other appropriate impact analyses sections (e.g., biology, water quality, air quality).

A "screening level" assessment means no supporting field data have been collected ahead of the proposed treatment to gauge ecological or human community endpoint risks; rather, risks are characterized *a priori* from the modeling of doses and potential effects to relevant receptors in Lake Davis and its surrounding ecosystem. Specifically, risks were screened by estimating chemical uptake (i.e., dose) in human and ecological receptor populations from the maximum estimated exposure point concentrations of rotenone formulation constituents expected from each complete exposure pathway. These estimated doses were then compared against published toxicity reference values (TRV) from the literature (for ecological populations) or site-specific health based screening levels (for human populations) for each significant formulation constituent. These comparisons were used to gauge whether the formulation constituents proposed for use presented a potential hazard to the receptor populations.

Screening level evaluations are intended to be conservative and are likely to overestimate potential exposures and associated risks. Using this type of screening approach is consistent with regulatory guidance for risk assessment, and addresses the objective of providing information useful for risk management decisions that protect human health and the environment. Actual risks may be much less than those discussed in this document. Monitoring to be conducted following treatment will be used to clarify how the projections of risk, as outlined in this appendix agree with measurements of water, air, and sediments collected after treatment. Significant deviations from projected risk, wherein risks are identified, will initiate adaptive management actions to reduce risks to acceptable levels.

J.2 PROBLEM FORMULATION

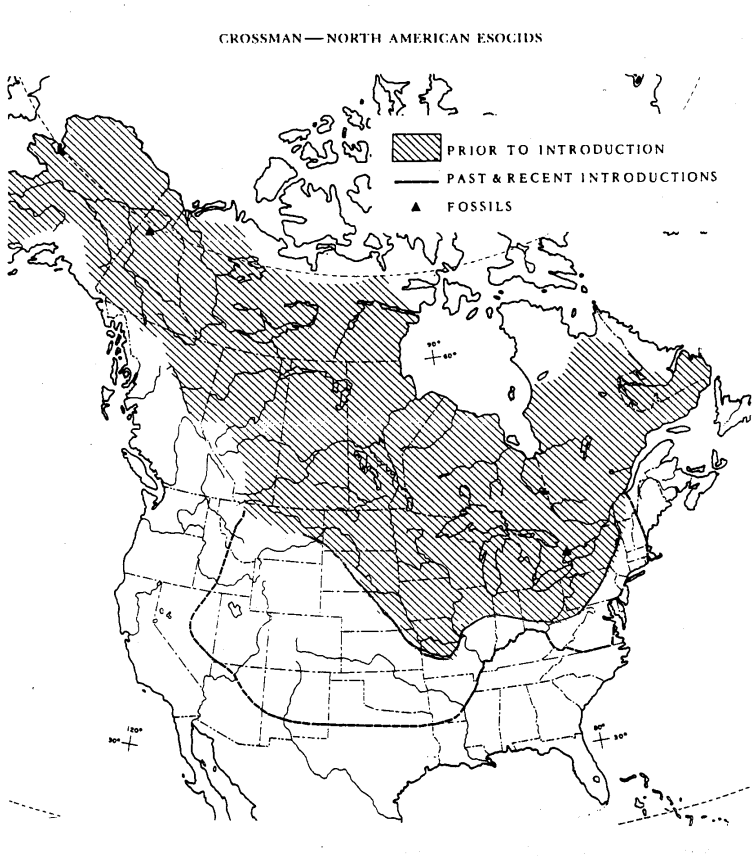
Problem formulation is the process in risk assessment where the scope of the problem to be addressed is defined, and the goals, objectives, hypotheses and methods for evaluating ecological and human health effects from the past or Proposed Project(s) are developed (USEPA 1998). Generally three 'products' are sought in the problem formulation phase: (1) risk assessment endpoints that adequately reflect management goals within the ecosystem under study, (2) conceptual site models that illustrate the key relationships between a "stressor" (i.e., the chemical(s) of potential concern) and the pathways by which the relevant receptors to the area(s) under study could be exposed, and (3) the analysis plan (i.e., methods) by which effects from the stressor(s) will be examined. To initiate the process, existing available information is reviewed from the project area to scope the problem, identify the receptors and potential exposure pathways of importance, and develop the approach for assessing risks from exposure.

J.2.1 Scope of Problem, Goals, and Objectives

Northern pike (*Esox lucius*), a predatory fish species in the Esocidae family with circumpolar distribution, is historically native to the upper midwest, northeast, and Canadian freshwaters. As demonstrated in Figure J-1, northern pike are non-native to the State of California and were illegally introduced into Lake Davis sometime before 1994, when they were first identified. From that initial introduction, the pike population rapidly expanded, impacting the local trout fishery and economy. In an attempt to eradicate the pike, the reservoir was treated with the commercially available rotenone formulation Nusyn-Noxfish® and the powdered rotenone product Pro-Noxfish® in October of 1997. While that treatment initially appeared successful, subsequent monitoring of the reservoir in May of 1999 revealed that a residual population of pike remained or was subsequently introduced again. Whatever the source of the pike introduction after the 1997 treatment, the current pike population is once again substantial, and its complete removal is a high priority for the DFG.

Pike introductions in other regions have demonstrated that pike have the potential to become the dominant fish species, preying upon and out-competing desirable sport and commercial fishes, as well as endemic aquatic species. Their ideal habitat, static or slow moving water with beds of aquatic macrophytes growing in shallow, clear water less than 39 feet deep in mesotrophic (medium productivity) to eutrophic (high productivity) waters (Casselman and Lewis, 1996) exists in many portions of California's central valley, affording the species extensive opportunity for expansion should it escape Lake Davis (Maniscalco 2002).

Lake Davis drains into Big Grizzly Creek which subsequently joins the Feather River, a major tributary of the Sacramento-San Joaquin watersheds. Should the pike escape or be moved from the reservoir, they could cause irreversible damage to the aquatic ecosystem and fisheries in the Bay-Delta estuary and its watershed, as well as potentially harm other areas of California and the western United States. Eliminating this threat is a high priority for the CALFED Bay-Delta Program Ecosystem Restoration Program, as outlined in their Strategic Plan (CALFED 1999, pg 461).



Source: Crossman 1978

Figure J-1 Northern Pike Distribution in North America

Since the current extent of northern pike in California is presumably limited to Lake Davis and its tributary streams, a temporary window of opportunity exists to eliminate the species from the state. The wider the distribution becomes, the less likely an effective eradication program can be successfully undertaken. Similarly, a wider geographical distribution of pike in California may also provide more opportunities to intentionally or unintentionally distribute this non-native predator throughout the western United States. Since February 2000, the DFG, in cooperation with a Steering Committee of local citizens, has been implementing a "control and containment" strategy as recommended in their Y2000 Plan.

Efforts at pike eradication since their reintroduction have resulted in about 55,000 pike being removed from the reservoir. However, data indicate that pike numbers continue to increase despite the control measures. For example, between 2000 and 2001 angler catches increased exponentially from 600 to 6,000. Based on the pike's rapid population growth, and the vulnerable ecological resources both within and downstream of Lake Davis, the DFG now considers pike eradication from Lake Davis and its tributaries an essential goal, with the associated objectives of protecting downstream aquatic resources, and restoring the important recreational trout fishery in the reservoir, in accordance with an overarching Fisheries Management Plan (DFG 2006, Appendix G).

J.2.2 Overview of Proposed Project and Alternatives

The rapid expansion of the pike population in Lake Davis prompted the DFG and the Steering Committee to consider all eradication options, leading to the final Proposed Project and treatment alternatives summarized in Table J-1. A detailed description of these alternatives, including the No Project alternative, is provided in Section 2 of the EIR/EIS. Given the nature of this technical appendix, only the Proposed Project and alternatives that involve the use of rotenone and neutralizing agents are described here (Alternatives A through D). Briefly, the DFG and the Steering Committee outlined the Proposed Project to meet the following objectives:

- be completed quickly
- use a method that has been proven to be effective in laboratory and field experiments
- use a method that is technically feasible to implement
- be in compliance with applicable laws
- be implemented in a manner that protects public health and safety
- minimize environmental impacts during and after application

As described in the Section 2.3.4 of the EIR/EIS in greater detail, each proposed treatment alternative may also require neutralization procedures with potassium permangante ($KMnO_4$) at a concentration of 4 mg/L-receiving water to ensure that no impacts from the rotenone treatment extend downstream below the neutralization zone in Big Grizzly Creek below the reservoir. Risks from neutralization with $KMnO_4$, as a chemically-based process, are also considered in this assessment. Four neutralization options are under consideration, as briefly summarized below:

1. **Reduce Flow and Pump Back Seepage**. All outflow from Lake Davis would be eliminated and dam seepage would be returned to the reservoir by pumps and pipes or tanker trucks. This option eliminates the risk of rotenone or potassium permanganate entering Grizzly Creek. All flow in a stretch of 150 yards directly below the dam would cease. Flow beyond the dry stretch would be provided by spring at about 60 gallons per minute.
2. **Offstream Neutralization of Minimal Flows**. Potassium permanganate would be mixed with lake water in baker tanks below the dam. Potassium permanganate-treated water would be passed through granular activated carbon (GAC) or some other substance to remove any excess $KMnO_4$ and then returned to the creek. Flows would be reduced to 0.2 to 0.5 cfs for 14 to 45 days in Grizzly Creek below the dam.
3. **Flow Releases of 1 to 2 cfs with Instream Treatment with KMnO4.** Flow from the dam would be curtailed for five days to allow the rotenone to mix in Lake Davis. Subsequently, 1 to 2 cfs would be released from the dam and treated in-stream with potassium permanganate.

Table J-1. Summary of Proposed Project and Alternatives that Involve Rotenone Use for Pike Removal from Lake Davis

Alternative Title	Proposed Project	Alternative A	Alternative B	Alternative C	Alternative D
	Lower Reservoir to 15,000 acre-feet and treat with liquid rotenone CFT Legumine® or Noxfish® formulation	**Lower reservoir to 15,000 acre-feet and treat reservoir with with powdered rotenone ProNoxfish® and tributaries with liquid rotenone Noxfish® formulation**	**Lower reservoir to 5,000 acre-feet and treat reservoir and tributaries with liquid rotenone Noxfish® or CFT® Legumine formulation**	**Lower reservoir to 35,000 acre-feet and treat reservoir and tributaries with liquid rotenone Noxfish® or CFT® Legumine formulation**	**Lower reservoir to 48,000 acre-feet and treat tributaries with liquid rotenone Noxfish® or CFT® Legumine formulation**
Lake Davis Reservoir Treatment Volumes in Acre Ft (FT) (or Liters L)	15,000 AF (1.94×10^{10} L)	15,000 AF (1.94×10^{10} L)	5,000 AF (6.46×10^{9} L)	35,000 AF (4.52×10^{10} L)	48,000 AF (6.20×10^{10} L)
Time to Refill	5 to 79 months	5 to 79 months	6 to 80 months	2 to 79 months	No refill required
Surface area of standing water (acres)	1,331	1,331	545	2,439	2,936
Amount of formulation used in reservoir	5,000 gallons CFT Legumine (18,905 L)	40,541 pounds ProNoxfish® powder	1,666.5 gallons Noxfish® (6,307.5 L)	11,667 gallons Noxfish® (44,159.5 L)	16,000 gallons Noxfish® (60,560 L)
Amount of formulation used in tributary streams	130 gallons CFT Legumine® or Noxfish® (492.1 L)	130 gallons Noxfish® (492.1 L)	137.5 gallons CFT Legumine® or Noxfish® (520.5 L)	115 gallons CFT Legumine® or Noxfish® (435.3 L)	100 gallons CFT Legumine® or Noxfish® (378.5 L)
Estimated # drip stations	27	27	35	24	21
Shoreline length (ft?)					
Surface elevation (ft)	4,749	4,749	5,738	5,759	5,764
Stream Length	34 miles	34 miles	38 miles	32	30 miles
Watercraft	1500 h	1500 h	1900 h	2500 h	2500 h
Vehicles (type)	6000 h	6000 h	6000 h	6000 h	6000 h

4. **Flow Release of 3 to 5 cfs with Instream Treatment.** Flow from the dam would be curtailed for five days to allow the rotenone to mix in Lake Davis. Water would be released from the dam at 3 to 5 cfs and neutralized in-stream with potassium permanganate as described in Option 3.

Under the four neutralization options proposed above, impacts from chemical risks would be greatest under Option 4, less so under Option 3, and essentially insignificant under Options 2 and 1, as these latter two options would either use no chemical, or would essentially contain the permanganate for a long enough period to ensure its complete degradation, filtration and/or chelation before its release downstream.

J.2.3 Project Area and Land Use

J.2.3.1 Project Area Location

Lake Davis is located in Plumas County, California, in the upper reaches of the Middle Fork Feather River watershed in the Sierra Nevada Mountains, at an elevation of 5,775 feet (Figure 2-3 in Section 2.2). A State Water Project reservoir, Lake Davis is operated by the California Department of Water Resources (DWR) and lies within the USFS Plumas National Forest. Lake Davis was impounded in 1966-68 by the construction of Grizzly Valley Dam on Big Grizzly Creek and is the product of inflow from Big Grizzly Creek, the largest tributary, and other lesser tributaries including Freeman and Cow creeks. When full, it has a surface area of 4,025 acres at a capacity of 84,371 acre-feet, a shoreline of over 32 linear miles, and an average depth of 21 feet. The deepest point of the lake is 108 feet, just upstream of the dam.

The EIR/EIS project area comprises the area directly affected by the proposed treatment and neutralization activities. This area includes Lake Davis, the waters draining into Lake Davis that may contain northern pike, and a portion of Big Grizzly Creek below Grizzly Valley Dam. The project area is represented by the watershed of Lake Davis and the portion of Big Grizzly Creek below the dam that flows to the Middle Fork Feather River, as shown on Figure 2-3, Project Area. Tributary streams within the watershed that are known to be flowing water areas would also be treated and are highlighted on Figure 2-3, along with proposed staging areas for both the reservoir and tributary treatments.

J.2.3.2 Land and Water Use in Project Area

The following two sections briefly describe the types of uses for the land surrounding Lake Davis that is also in the project area. The uses of the water in the project area are also summarized.

J.2.3.2.1 Land Use

The area immediately surrounding the lake is zoned for recreational use and boating, shoreline-based camping, hiking, wildlife viewing, and associated day-uses are popular activities (Rischbieter and Nicholas, 2002; Hinton and Nicholas, 2004). There are 185 seasonal campsites at the three reservoir campgrounds and one children's camp (at

Walton's Grizzly Lodge) outside the project area located adjacent to the Ice Pond, with another (Grizzly Creek Ranch) farther down Big Grizzly Creek that is operated year-round. In the autumn months, when the majority of the Proposed Project is planned, water related uses will not be as active as in the summer. Water and air temperatures are lower in the fall, and will likely be less attractive for water sports during that season. Hunters and anglers, however, may still use the reservoir and its environs for their activities.

At the southern end of the reservoir, part of the area is zoned for residential and industrial use. A portion of the Big Grizzly Creek watershed area into which the reservoir drains is also part of the Project Area. Land use zoning designations for this area are rural, rural with agricultural buffers, suburban, and secondary suburban (DFG, undated).

The forested areas immediately surrounding Lake Davis are designated in the Plumas National Forest Land Resource Management Plan as "Important Timber." Interspersed within this area are also other areas designated as "Timberland Production Zone." Given that the project duration is considered temporary and that the DFG will coordinate with Plumas National Forest to assure that the Proposed Project activities do not conflict with logging (DFG 2005d, Appendix B).

J.2.3.2.2 Water Uses

Lake Davis is located within the Plumas National Forest, is designated as a "special water area" in the Plumas County General Plan, and supports an important trout fishery managed by the California Department of Fish and Game (DFG). In addition to its important recreational and fishery roles to the local economies of the City of Portola and Plumas County, Lake Davis water is also used for irrigation and has been developed as a source of domestic water for the City of Portola and the Grizzly Lake Resort Improvement District (GLRID). Although developed as a drinking water source, the reservoir is not currently used in that manner, since the new Plumas County Water Treatment Plant is not currently operating. If 'on line' by the time of the proposed treatment in 2007, it will be turned off until after the selected treatment is completed, and post treatment monitoring by DHS confirms the safety of the water supply for public drinking water consumption. Thus, the reservoir water would not be used for drinking by humans during the period when rotenone treatment and neutralization activities associated with the Proposed Project could impact water quality.

J.2.4 Management Goals and Assessment Endpoints for Estimating Risk

J.2.4.1 Ecological Health

The management goal of this ecological risk assessment is to protect the environment and non-target ecological receptor populations from avoidable risks of injury from the proposed use of rotenone in Lake Davis. This goal is consistent with the regulatory goals of the federal Toxic Substances Control Act (TSCA §2[b][1], Clean Water Act (304(a)CWA), the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), state policy of the California Water Quality Control Board,, and the management goals of the DFG as outlined in California Fish and Game Code sections 1700 and 5501. The assessment endpoint from which risk will be

characterized is the estimated survival of potentially exposed ecological receptor populations known to utilize the project area where rotenone treatment is planned and/or could be distributed.

J.2.4.2 Human Health

The management goal for the human health portion of this assessment is to protect human populations in the general public from injurious exposure to rotenone formulation constituents during the proposed treatment by complying with all applicable and relevant regulatory standards, label use requirements, and site safety and health plan specifications. The human health risk assessment is not designed to and will not evaluate potential applicaton worker exposures or other applicator associated exposure condiserations. The potential exposures and use of protective equipment for those workers is addressed separately by the label use restrictions and a project specific health and safety plan.

When applied to surface waters, the following use restriction goals are also sought: (1) prevent the human consumption of fish killed by the rotenone treatment, (2) prevent the use of the treated water for irrigation purposes, and (3) prevent the release of the treated waters within one-half mile of a drinking water and/or irrigation water intake line. Estimated cancer and noncancer hazards from potential exposure to constituents in the rotenone formulations will be used as the assessment endpoints used to characterize risks to potentially exposed human populations in the project area.

J.2.5 Conceptual Model and Risk Hypothesis

A conceptual site model (CSM) is used to represent the potentially complete exposure pathways that ecological and human ‘receptors’ in the chemically treated environment could encounter. A typical CSM outlines: (1) all potential sources of chemicals associated with a chemical application or remediation project area; (2) release and potential chemical migration mechanisms; and (3) potential exposure pathways, including receptors, which lead to contact with site related constituents at an exposure point.

Based on the description of the project action and alternatives, the primary chemical exposure source can be considered the intentional release of rotenone formulations into the Lake Davis reservoir, with the secondary source the spraying of formulation along the edge of the lake and tributary streams inaccessible to boats. Once released, the primary routes for formulation constituents to distribute into the environment would include:

- dissolution into lake surface water
- adsorption onto lake sediments
- adsorption onto aquatic and riparian vegetation
- volatilization into air of formulation constituents from the surface of the treated waters, and subsequent dispersion in air

Thus, the ‘exposure points’ through which non-target ecological and/or human receptors could contact or otherwise receive “doses” of rotenone formulation constituents include:

(1) via treated surface water, (2) via treated vegetation, (3) via sediment contact and/or ingestion, (4) via groundwater used for drinking that is potentially contaminated by formulation constituents, (5) via bioaccumulation from dead target organisms (i.e., fish), and (6) via inhalation of volatilized drift. Based on these release mechanisms, risks to aquatic, terrestrial, and human health receptors are potentially significant, and the null and alternative hypotheses presumed for this screening level assessment can be stated as follows:

- Ho: rotenone application at 1 mg-formulation/L-receiving water (50 μg-rotenone/L), the label requirement, will yield significant toxicologically-based impacts to non-target aquatic and terrestrial biota, and human health endpoints.
- Ha: rotenone application at 1 mg-formulation/L-receiving water (50 μg-rotenone/L), the label requirement, will not yield significant toxicologically-based impacts to non-target aquatic and terrestrial biota, and human health endpoints.

J.2.5.1 Potential Ecological Receptors

Lake Davis lies within a broad valley that includes riparian, wetland, grassy meadow, big sagebrush, and pine-dominated habitats that support a variety of wildlife, including special status species. Over 170 species of birds, 69 species of mammals, 14 species of reptiles, and 8 species of amphibians have been documented, as detailed in Section 7 of the EIR/EIS. The most common amphibians and reptiles include Pacific treefrog (*Hyla regilla*), western toad (*Bufo boreas*), long-toed salamander (*Ambystoma macrodactylum*) and common garter snake (*Thamnophis sirtalis*). Typical mammal species include at least seven species of bats, shrews (*Sorex* spp.), moles (*Scapanus* spp.), mice (*Peromyscus* spp.), pocket gophers (*Thomomys* spp.), western gray and Douglas squirrels (*Sciurus griseus, Tamiasciurus douglassii*), American beaver (*Castor canadensis*), mountain beaver (*Aplodontia rufa*), cottontail rabbit (*Sylvilagus nuttallii*), raccoon (*Procyon lotor*), coyote (*Canis latrans*), and mountain lion (*Felis concolor*). Two species of big game wildlife are found within the Lake Davis area: mule deer (*Odocoileus hemionus*) and black bear (*Ursus americanus*). The entire Lake Davis shoreline and surrounding forest constitutes deer summer range and is also used by bears.

In addition to these common species, a total of 22 special status wildlife species, those with either federal, state, USFS, or Calfed conservation strategy listings have been identified with the potential to occur in the project area (see Section 7). Only one species, the threatened bald eagle, is federally listed, although it is considered endangered by the state.

Obligate aquatic animal species in the Lake Davis project area include the array of 22 different fish species that currently use Lake Davis and its tributaries, and an extensive list of aquatic macroinvertebrates and zooplankton, as summarized in Section 7 of the Draft EIR/EIS. Lake Davis zooplankton include cladocerans (e.g., *Daphnia pulex, Diaphanosoma* spp., *Ceriodaphnia* spp. & *Bosmina* spp.), rotifers (*Asplancha* spp.), and copepods in the orders Calanoida and Cyclopoida. One special status aquatic invertebrate species has been found in the Project Area. The amphibious caddisfly (*Desmona bethula*) has been conclusively identified in the project area. The amphibious caddisly is endemicto the Sierra Nevada. The aquatic larvae dwell in low order streams in open, wet-meadow areas (Erman and Nagano 1992). Like many other caddisflies (Order: Trichoptera), *D. bethula* larvae build

cases of sand and organic debris. Larvae pupate by the late summer or early fall and emerge as winged adults in early October.

Four caddisflies listed as California Species of Concern could potentially occur within the Project Area, based on their described geographic distribution. These species are currently not believed to be present in the Project Area. These species include:

Golden-Horned Caddisfly (Neothremma genella)

This species lives in second or sometimes first order streams in the Sierra Nevada over a wide range of elevations (Erman and Nagano 1992). It has been identified in Madera, Plumas, and Sierra counties. Larvae live on rocks in fast water and build horn shaped cases of sand and silk. Adults emerge from mid-August to early October. *N. genella* is easily confused with *Farula praelonga* (CNDDB 2006).

Sagehen Creek Goeracean Caddisfly (Goeracea oregona)

This species is known from several locations in both California and Oregon. Larvae live on rocks in relatively warm (48.2°F to 51.8°F [9°C to 11°C]) springs where they feed on vegetation and may take two years to complete their life cycle. Adults have a long emergence period (June to October) when they exhibit almost flightless mating behavior (Erman 1998).

Long-Tailed Caddisfly (Farula praelonga)

The larvae of this species live in first and second order spring streams in the Sierra Nevada, in shaded areas with constant (around 48.2°F [9°C]) temperatures. The larvae of the *Farula* genus build slender cases of fine sand and silk and graze on diatoms on the surfaces of rocks. The larvae pupate in aggregations on the underside of rocks. *F. praelonga* is easily mistaken for *N. genella* (CNDDB 2006).

King's Creek Ecclisomyian Caddisfly (Ecclisomyia bilera)

This species has been identified in Lassen County, Sierra County, and other sites in the northern Sierra Nevada. The larvae live in small, cold springs among rocks and gravel where they construct straight slender cases and probably feed on algal and plant material (CNDDB 2006). Adults emerge from May through August and exhibit near flightless mating behavior (Erman 1998).

Springsnales

Specimens of snail from the Family Hydrobiidae have been collected from springs and streams within the project area. Gilled springs nales spend their entire life cycle in spring waters feeding on algal and plant material. They spawn only once in their life. Refer to Section 7.1.1.5 of the Draft EIR/EIS for a more detailed discussion of aquatic species.

Plants in the project area where rotenone and other measured formulation constituents in CFT Legumine or Noxfish could be used are summarized in Section 7 of the Draft EIR/EIS,

and are consistent with the riparian, wetland, upland, and scrub-shrub mosaic of habitats found in northern portions of the Sierra Nevada. Toxicity data have not been identified to suggest that plants could be adversely affected by the rotenone treatment or formulation constituents; this finding is consistent with the recent conclusions of the USEPA when evaluating rotenone for registration (USEPA 2005). This conclusion is also supported by empirical evidence from numerous treatments with rotenone for fish management purposes, and by the use of rotenone as a common pest management tool applied directly to garden vegetation at much higher application rates than would be expected from the aquatic application proposed. Given that any terrestrial plant exposure would be inadvertent to the water treatment, exposure to terrestrial plants is expected to be insignificant.

As opposed to modeling exposure and risk to all of the possible non-target species that could occur in the Lake Davis project area summarized above, surrogate species of ecological receptor "guilds" were examined. Guilds are species groups with similar life histories or niches in the environment. Surrogate species within guilds can be used to estimate exposure rather than estimating exposure for each individual species where a chemical could be applied. The assumption of this approach is that the general characteristics of each guild will provide risk estimates that are representative of the entire guild. As such, each guild can be extrapolated more broadly than single species estimates. The underlying concept is that each receptor falls into a group of potential receptors that function in similar ecological niches or "guilds." A single surrogate, such as the great blue heron, for which reliable life-history information is available, may be used for calculating risk and the results may then be extrapolated to the guild as a whole that might include a special status species such as a white pelican. This approach allows the risk assessment to directly evaluate species for which the best exposure information is available. This approach also allows results to be extrapolated to a broader range of potential receptors, thereby maximizing data usage and applicability of results.

Figure J-2 illustrates potential exposure pathways to the ecological receptor guilds of relevance to the Lake Davis area. Several of these species are under the special status list, as summarized in Section 7. In brief, exposures are possible through ingestion, dermal contact, and inhalation routes. When a rotenone formulation is used as a piscicide, as proposed for this project action, it will be diluted directly into the receiving water—in this case Lake Davis and its tributaries. Once distributed into the water, non-target receptors—essentially all aquatic non-pike organisms resident to the treatment waters will likely receive some level of exposure. In addition to these direct pathways for exposure, the potential for bioaccumulation must be considered as an indirect pathway, particularly for terrestrial ecological receptors such as fish eating birds and mammals.

Whether the route of exposure is complete depends on the receptors' habitat and life histories as associated with the project action. Figure J-2 attempts to reflect how these differences are seen among the short list of receptor guilds modeled. Closed squares indicate complete exposure pathways for receptors of interest. Open squares indicate incomplete exposure pathways. Closed circles represent potentially complete exposure pathways for which dosages received are likely insignificant. As demonstrated, direct contact exposure with the treated water is considered a complete pathway for all aquatic organisms, amphibians, and reptiles where the assumed route of uptake is through bioconcentration from the water.

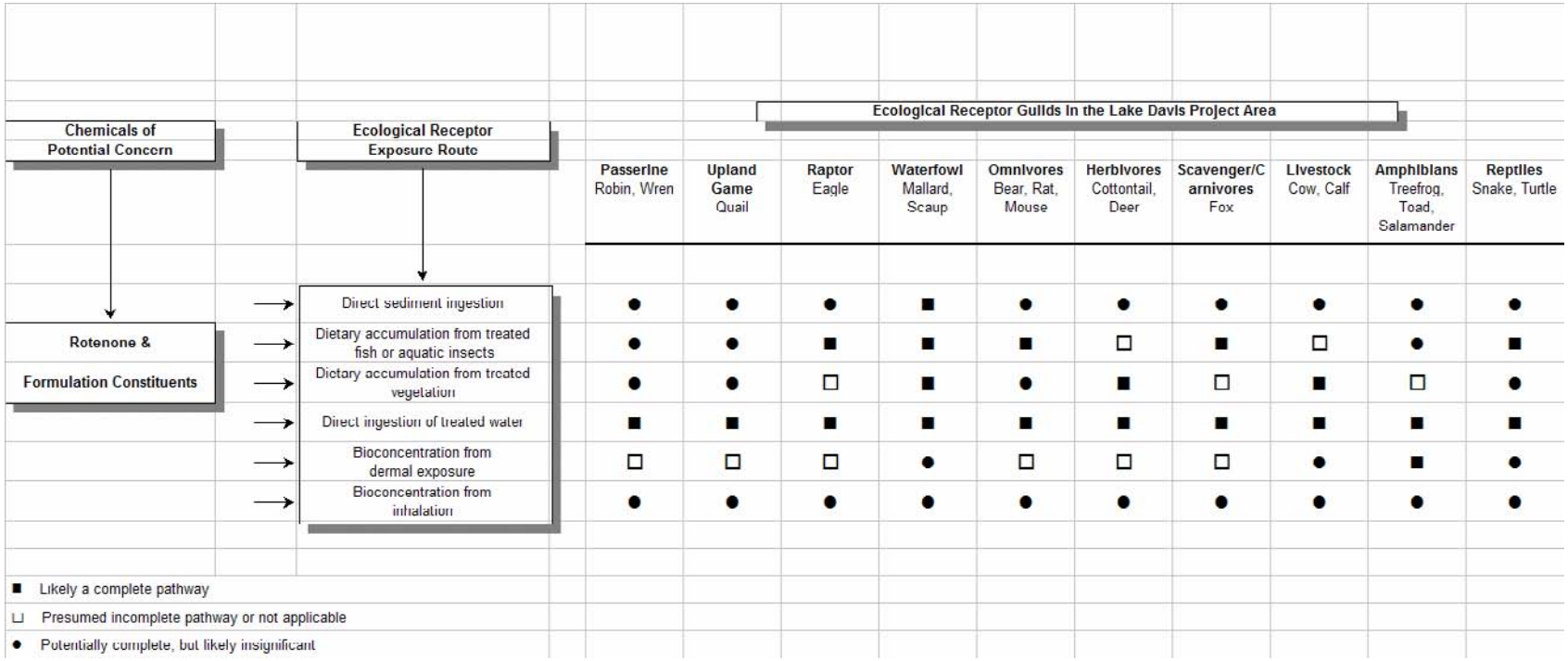


Figure J-2 Ecological Receptor Conceptual Site Model

Exposure of terrestrial biota through dermal contact is likely complete, but considered insignificant because of skin barriers and the expected minimal skin contact that would likely occur during the abbreviated treatment period (when excessive activity around the lake will discourage, (but not prevent) the use of the area by terrestrial ecological receptors). Inhalation exposure is similarly considered complete, but likely insignificant for the same reasons. Ingestion exposure through drinking water and food is considered complete for terrestrial biota.

J.2.5.2 Potential Human Receptor Populations

Based on the land use and activities described in Section 2.3.2, the human receptor populations that are members of the general public, with some potential for exposure were separated into two main groups, (1) those with the potential to be present in the immediate Lake Davis treatment area during the months identified for the project—the Project Area Populations, and (2) those expected to be nearby, but not likely in the immediate area of treatment.

J.2.5.2.1 Project Area Human Populations Potentially at Risk of Significant Exposure

The following human populations engaged in associated activities would likely be present during the months identified for the project:

- **Water Sports Enthusiasts.** This includes the boaters, swimmers, water skiers, wind surfers, and others interacting directly with the water in Lake Davis
- **Hunters.** Although the margin of forest around the lake that is to be treated is small, it is possible that hunters may be present
- **Fishermen.** Anglers may be fishing from the shore as well as from boats
- **Campers.** Some of the campground areas may have overlap within the treatment area for the project.
- **Pesticide Applicators.** Those workers who are employed by, or contracted by, DFG to apply the piscicide formulation to the project area will be present.

Human exposure to rotenone and other chemicals in the commercial formulations proposed for northern pike eradication in Lake Davis may result in adverse health effects depending on the amount (i.e., concentration) of chemical contacted, the duration of exposure, and the manner by which humans contact the chemical(s) (e.g., breathing, touching, swallowing). Many of the chemicals in the piscicide formulations, discussed in Section 3, including rotenone, breakdown quickly in the environment and will not remain in amounts or forms that result in health concerns. Further, as indicated above, workers applying the pesticide application to the lake and other designated areas will take safety precautions including wearing protective clothing as defined in a site specific health and safety plan. However, there is a potential impact to the general public if they are present during and immediately following the actual piscicide application and are not wearing appropriate protective clothing.

It is therefore assumed that institutional controls (forest closure) will be used to prevent public exposure to the piscicide formulation during and following application.

Appropriate institutional controls for such broad scale chemical treatments could include the closing of the treatment area to water sports, hunting, fishing, camping, and any other recreational activities during and after piscicide application. Forest Closure #2 is designed for this purpose and is part of all treatment alternatives. For those in the nearby areas, contact with rotenone formulation constituents will not likely be a concern for health. However, information indicating that contact should be avoided should be disseminated to those locations and individuals or businesses that may have access to the treatment area.

Implementing the institutional controls identified above would eliminate the presence of hunters, anglers, and water sports enthusiasts from the project area. However, some individuals may not speak or read the language of the information distributed, and there may be occasional contact by individuals whom we will call "unauthorized visitors," since the area will be officially closed. Therefore, this additional potential receptor will be considered:

- **Unauthorized Youth.** It will be assumed that this individual is a youth between the ages of 14 and 18, since that is a more conservative evaluation than an adult. They are assumed to camp in the treatment area and use the lake for water recreation.

J.2.5.2.2 Nearby Area Human Populations at Potential Risk of Exposure

There are some residences near but not within the treatment area. There are also a few businesses such as the general store that is near the treatment area, and employees of those businesses could be present during the duration of the project. There is a children's camp downstream from the treatment area along Grizzly Creek and adjacent to the Ice Pond at Walton's Grizzly Lodge. A second children's camp is for disabled children (Grizzly Creek Ranch) and is located farther away from the treatment area downstream on Big Grizzly Creek. The first camp is active during the summer and through August, which covers a portion of the proposed project time. Therefore, the following additional receptors will be retained for further human health risk evaluation of 'nearby receptors':

- **Nearby Residents.** Residents are assumed to continue to live in their homes, but that they do not go into the treatment area or contact the lake or other surface water or revealed lake sediments due to the drawdown of the lake. Groundwater is assumed to be the source of potable water, not surface water from the lake or creeks.
- **Nearby Workers.** Workers are assumed to conduct their usual work activities, but that they do not actually go into the treatment area or contact the lake or other surface water or revealed lake sediments.
- **Recreational Child Camper.** Although the treatment area does not extend to the camp location or the Ice Pond, it is possible that chemicals may accidentally reach the Ice Pond, so this receptor is included to be conservative and address unplanned contingencies.

Figure J-3 shows the CSM for the potential human populations that may be present during and following the piscicide application, as well as, the anticipated potentially complete exposure pathways for each receptor group. The symbols used for this CSM are as follows:

- **Solid (Closed) Circle.** Represents potentially complete exposure pathways that are likely to contribute the majority of the total exposure, and
- **Open Box.** Represents incomplete exposure pathways.

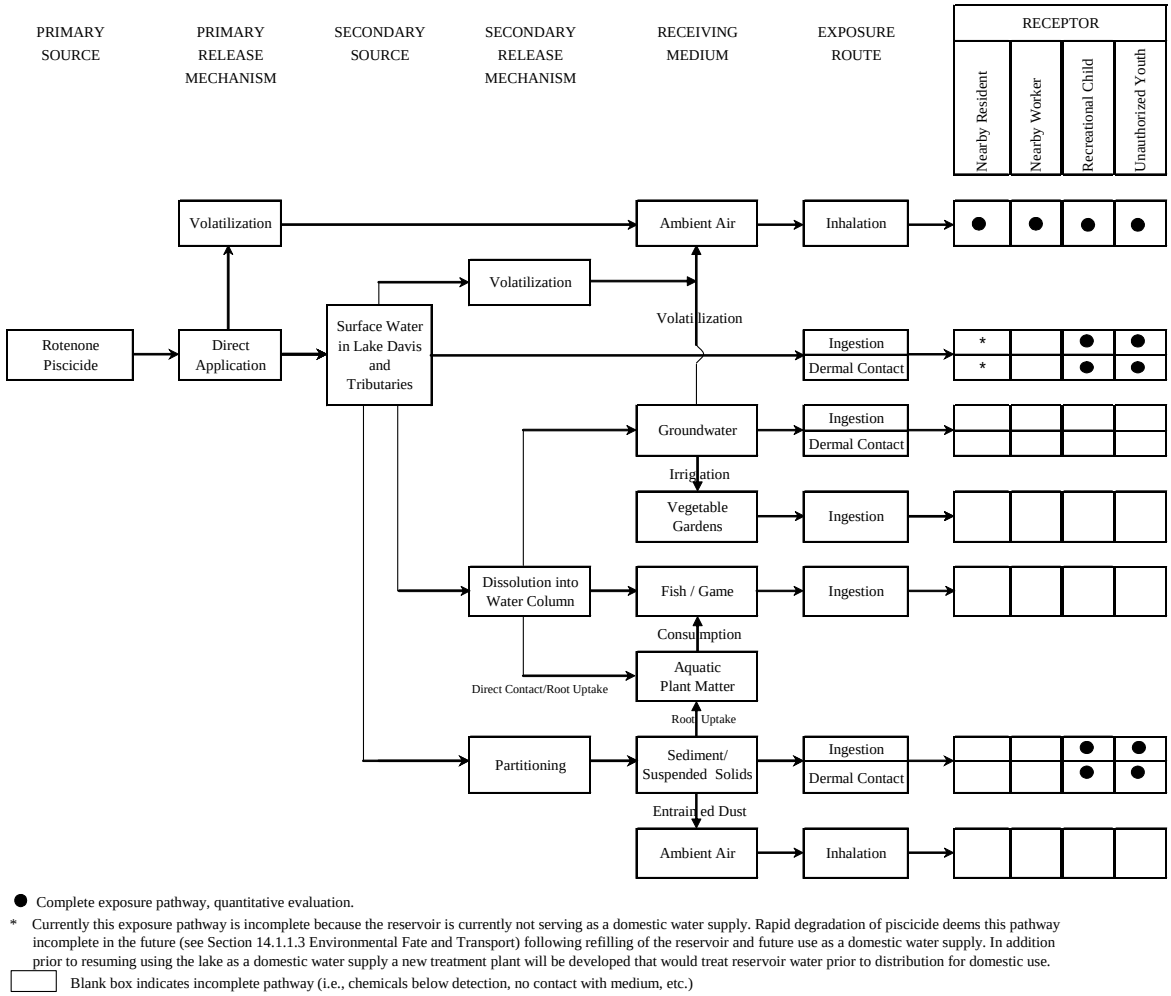


Figure J-3 Conceptual Site Model for Human Exposure

As a result of chemical migration into these media, the risk assessment will evaluate the magnitude of exposure by estimating:

- Inhalation exposure to volatile constituents of the piscide formulation by a nearby resident, nearby worker, recreational child and unauthorized unauthorized youth, and
- Ingestion and dermal contact with lake surface water and sediment by a recreational child and unauthorized unauthorized youth

J.2.5.2.3 Insignificant Exposure Pathways to Human Health

Groundwater exposure and all associated direct (i.e., ingestion) and indirect (i.e., ingestion of irrigated farm produce) exposure routes to groundwater were deemed incomplete exposure pathways because historically groundwater has not been impacted by rotenone use for piscicidal purposes (Carlson, 1999). One hydrogeologic study suggested that there is a small possibility of some hydraulic connectivity between Lake Davis and wells downstream of Grizzly Dam along Grizzly Road. However, most of the aquifer is likely recharged by the aquifer underlying Crocker Mountain and through snowpack and rainfall (DFG 2005). Long-term groundwater monitoring studies were conducted to address this concern following the previous application of rotenone to Lake Davis. Following the last application, 5 domestic wells ranging in depth of 85 to 239 feet and located 1,500 to 2,500 feet downgradient of the lake were sampled 5, 14, 90, 194, and 324 days after the 1997 application (Finalyson et al., 2001). All samples were analyzed for rotenone, rotenolone, VOC, and SVOCs with analytical methods approved for drinking water standards and no residues of rotenone or rotenolone were found in any of the wells monitored or any of the VOC and SVOCs (Finlayson et al., 2001).

Of similar note, the Plumas County Environmental Health (PCEH) department is responsible for overseeing a 10-year groundwater monitoring study which monitors over 80 wells in the Lake Davis community. Recently the PCEH has completed the seventh year of groundwater monitoring and since 1999, no piscicide or any primary piscicide component has been detected in any of the wells. Several volatile organics were detected at low levels; however, none of the compounds exceeded the Maximum Contaminant Level (MCL) allowable for drinking water and these compounds are commonly found in most households (PCEH 2006). As a result of these studies and the analysis in Sections 4.2.4 and 14.2.4.2 of the EIR/EIS, the risk assessment deemed this pathway incomplete and it was not evaluated further.

Other pathways that were deemed insignificant exposure pathways for human health risks included the ingestion of contaminated fish and game inhabiting the lake following the completion of piscicide application activities, and inhalation of sediment as particulate matter following the lake drawdown. As part of the protocol for conducting fish eradication activities, fish are not stocked into a treated area until all of the toxic effects are gone and rotenone has dissipated. Hence, newly stocked fish will not accumulate residues of rotenone from the water. Residues of rotenone in tolerant fish that survive a rotenone treatment won't last for more than several days because the bioaccumulation potential for rotenone is low and the rapid metabolism of the chemical results in a half-life of about 1 day (Gingerich and Rach 1985; Gingerich, 1986 *as cited in* Finlayson et al, 2000). Fish that are killed as a result of the application of piscicide will be collected according to the fish removal and disposal plan described in Section 2.3.6. The unauthorized visitor is the only receptor that has the potential for contact with dead fish remaining after treatment is complete. If dead fish from the site were consumed, the primary health concern would be the acute illness associated with poisioning from consuming *Salmonella sp*. and other bacteriological species likely to be present in the flesh of fish that have been dead for a while (Finlayson, 2000). Since there is a strong foul odor from dead fish that would prevent accidental consumption, it is unlikely that such dead fish will be consumed by the unauthorized visitor. Therefore, this pathway is also considered insignificant and likely incomplete.

With respect to suspension of rotenone contaminated sediments as dust during lake drawdown, this pathway would also be insignificant, for two reasons: (1) rotenone will be primarily be applied to water, with only a small amount of exposed sediment being treated (Personal communication between W. Curley and B. Finlayson, 2006), and (2) rotenone on surfaces exposed to air and sunlight is not stable and degrades rapidly. For example, the half-life of rotenone on the surface of bean leaves is 1.4 to 2.9 hours. Since the wet sediments would first have to be dried prior to dust formation, rotenone would not be expected to be present after the time that it would take for the sediments to dry sufficiently to form dust. Finally, there would not be exposure to plants growing in affected lake sediments since aquatic plants are not a food source to human receptors.

J.2.6 Analysis Plan for Characterizing Risks to Ecological and Human Health from the Proposed Project and Alternatives

This section outlines the specific methods that are employed to characterize the risks to the ecological and human receptor populations identified in the conceptual model to have potentially significant exposure to rotenone or rotenone formulation constituents.

J.2.6.1 Ecological Toxicity Risk Assessment Methods

The approach used in this ecological risk assessment borrows from federal guidance for conducting ecological risk assessments (USEPA 1998), and state guidance (Cal/EPA 1996). Briefly, the approach involves:

- identification of chemicals of potential concern (COPCs),
- selection of toxicity reference values (TRVs) for the COPCs
- identification of habitats, biological communities, and biological receptors of potential concern where exposure to COPCs could occur
- identification of exposure parameters and appropriate uptake equations,
- prediction of estimated exposure to COPCs, and
- comparison of estimated exposure to recognized toxicological hazards associated with the COPCs to ascertain risks.

Identification of the COPCs and TRVs are summarized in the Hazard Assessment portion of this appendix (J.3), following the review of the literature on the substances that could be released from the rotenone treatment.

Habitats where exposure might be significant to ecological receptors were previously identified in the conceptual model, including all the treatment areas summarized in Table J-1, and the broader project area identified in Figure 2-3. Given the intentional aquatic application, aquatic receptors sensitive face the greatest risk of exposure to rotenone and rotenone formulation constituents.

Exposure is calculated from the general equation [I]

$$[I] \qquad \text{Daily intake} = CM * CR * FI * AF/BW$$

Where,

BW = Body Weight

CM = Concentration of contaminant in exposure media(s) of concern.

CR = Contact Rate—The estimate of the quantity of the medium consumed per day.

FI = Fractional Intake—The fraction of time (site use factor) spent in contact with the contaminated media (e.g., the proportion of the total diet obtained from the site, as extrapolated from information such as home range data on the species, or empirical findings).

AF = Absorption Fraction—The amount of contaminant contacted (e.g., consumed) that is actually assimilated into tissue to assert a potentially toxic effect.

Recognizing that the contact rate may represent the additive uptake by several pathways (e.g., ingestion of animal and sediment matter treated with rotenone) requires the estimate of the additional dose from other exposure media. These modifications, along with the input parameters necessary to gauge dose to the array of ecological receptors modeled, are detailed in the Exposure Assessment portion of this appendix (J.4).

The Hazard Quotient (HQ) is then calculated to characterize risks from the estimated exposure doses by dividing the dose received, by the chronic or acute toxicity reference value—whichever was available from the literature. For obligate aquatic species, risks were characterized by using the estimated concentration of rotenone formulation constituent with complete mixing as the Exposure Point Concentration (EPC) and dividing that by the effect concentrations identified in the literature, as identified in [II]

$$[II] \qquad HQ_1 = EPC/TRV$$

Where:

EPC = Exposure Point Concentration (i.e., the concentration of contaminant in the exposure media), and

TRV = Toxicity Reference Value, as summarized by species in section 3 of this report.

The calculation of HQs, by species, represent the culmination of the exposure and toxicity assessments, and these metrics are provided in the Risk Characterization chapter of this appendix (J.5).

J.2.6.2 Human Health Toxicity Assessment Methods

Methods for assessing possible human health toxicity also follow standard regulatory guidance (USEPA 1989; Cal/EPA, 1992 and 1999). Toxic responses in humans are categorized and evaluated for two groups, carcinogenic responses, and non-carcinogenic responses. Chemicals that demonstrate evidence of carcinogenicity are referred to as carcinogens. Excessive exposure to all COPCs can produce adverse noncancer health effects while the potential for causing cancer is limited to carcinogens. Therefore, all COPCs in the rotenone formulations will be evaluated for potential noncarcinogenic effects, while potential cancer effects are evaluated only for carcinogens. The noncancer toxicity values are termed

reference doses (RfDs), and the cancer toxicity values are termed cancer slope factors (SFs). These values will be reviewed from a hierarchy of information sources as follows:

1. SFs or RfDs promulgated in California regulations
2. SFs or RfDs used to develop environmental criteria promulgated in California regulations.
3. USEPA's Integrated Risk Information System (IRIS). Access to this database can be obtained through the National Library of Medicine's "TOXNET" system, (301) 496-6531; USEPA's Risk Information Hotline, (513) 569-7254; or a variety of commercially available databases.
4. The most current edition of USEPA's Health Effects Assessment Summary Tables (HEAST).

The toxicity values used for human health risk assessment are determined by the State of California Office of Environmental Health Hazard Assessment (OEHHA), when available, and by the USEPA as the next source of information. These government agencies convene panels of scientific experts in toxicology, medicine, exposure, and other relevant disciplines to review the available scientific literature for specific chemicals and then determine acceptable regulatory toxicity values. The toxicity values used in this evaluation were all taken from these established regulatory sources of information.

J.2.6.2.1 Evaluation of Potential Carcinogenic Risks

To assess potential human carcinogenic risks from rotenone and constituents in the proposed rotenone formulations, a two-part evaluation will be used: (1) determination of a weight-of-evidence (WoE) classification, and (2) calculation of a cancer slope factor (SF).

Weight of Evidence (WoE) for Classification of Carcinogens

The WoE classification is determined by the scientific panels that review toxicity literature for OEHHA and USEPA as described above. The WoE classification reflects an evaluation of the amount of data available that can be used to classify a constituent as a human carcinogen. Data used to determine the WoE consist of epidemiological data and the results of animal tests. Based on USEPA's 1986 "Proposed Guidelines for Carcinogen Risk Assessment," six weight-of-evidence categories exist:

A	Human carcinogen (sufficient evidence of carcinogenicity in humans)
B1	Probable human carcinogen (limited human data are available)
B2	Probable human carcinogen (sufficient evidence in animals and inadequate or no evidence in humans)
C	Possible human carcinogen (limited evidence of carcinogenicity in animals)
D	Not classifiable as to human carcinogenicity
E	Evidence of noncarcinogenicity for humans

The carcinogen classification of the formulation constituents under consideration is discussed in Section J.3 (see Table J-14).

Estimation of Carcinogen Potency (SF) and Unit Risks (UR)

The potency of carcinogens can be expressed in several ways, including the cancer slope factor (SF) and the "unit risk" (UR). The SF is a plausible upperbound estimate of the probability of a carcinogenic response per unit intake of a constituent over a lifetime. The SF is usually the UCL_{95} of the mean slope of the dose-response curve and is expressed as inverse milligrams per kilogram per day [$(mg/kg/day)^{-1}$]. Toxicity values for carcinogenic effects can also be expressed as risk per unit concentration of the substance in the medium of exposure, referred to as a unit risk (UR). The methods used by USEPA to derive SFs or URs are described in RAGS, Part A (USEPA 1989). For carcinogens, USEPA usually assumes a nonthreshold response, i.e., at every dose level of a carcinogen there is some amount of adverse response. In other words, no dose is believed to be risk-free. However, recent studies suggest that some potential carcinogens may have a threshold dose. Currently, USEPA does not consider any of the COPCs to exhibit a threshold dose for carcinogenicity; therefore, potential thresholds for carcinogenic effects won't be considered in this RA.

J.2.6.2.2 Evaluation of Noncarcinogenic Effects

The toxicity values used in the human health risk assessment to estimate the potential for adverse noncancer health effects are termed reference doses, or RfDs. An RfD is an estimate (with uncertainty spanning approximately an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects if experienced continuously during a lifetime and is the toxicity value most often used to evaluate the noncarcinogenic impacts from exposure to constituents.

The RfDs are specific to the route of exposure (e.g., an inhalation RfD is used for inhalation exposure), critical effect (developmental or systemic), and the length of exposure evaluated. Chronic RfDs are specifically developed to be protective against long-term exposure to a constituent. Subchronic RfDs are developed to characterize potential noncarcinogenic effects associated with short-term exposures. The derivation procedure for an RfD can be found in RAGS Part A (USEPA, 1989) or other technical guidance documents for criteria development.

J.3 TOXICITY ASSESSMENT

This section of the risk assessment reviews the toxicological literature on rotenone and the most concentrated formulation constituents in order to identify the most appropriate toxicity reference values (TRVs) from which to characterize ecological receptor risks and the most appropriate subchronic health based screening levels (HBSLs) from which to characterize risks to exposed human populations in and near the project area. This section also summarizes the fate, transport and persistence of the formulation constituents that could be anticipated in order to qualitatively assess the potential for longer term environmental exposures to formulation constituents or their breakdown products.

J.3.1 Rotenone Origin, Synthesis and Uses

Rotenone ({2R,6aS,12aS}-1,2,6,6a,12,12a-hexahydro-2-isopropenyl-8,9-dimethoxychromeno[3,4-b]furo[2,3-h]chromen-6-one) is a naturally occurring flavonoid derived from the roots of tropical plants in the pea and bean family (*Leguminosae*), including jewel vine (*Derris* spp.) and lacepod (*Lonchocarpus* spp.) found in Australia, Oceania, southern Asia, and South America (Finlayson *et al.*, 2000 cited in USEPA, 2005). Resins extracted from these plants' roots with ether or acetone may contain between 2 and 40% rotenone (Ray, 1991). Rotenone is a non-specific botanical insecticide, acaricide, and piscicide and was historically utilized by indigenous tribes in South America and Malaysia as an effective method to catch fish for food. Roots containing the compound were ground up and the resulting pulp was added to water bodies containing fish, killing them in minutes or hours.

The use of rotenone as a pesticide was first patented in Britain in 1912. Today, rotenone's natural origin, its high toxicity to many pest organisms, relatively low toxicity to birds and mammals, rapid detoxification in warm water, and lack of environmental persistence has made it a popular and effective organic pest management tool for gardeners, for lice and tick control on pets, and for modern fish eradication projects as part of water body management (USEPA, 2006). In the United States rotenone is classified as a General Use Pesticide (GUP) although uses on cranberries and for fish control are restricted (Extoxnet 1996).

Rotenone is either extracted from plants and formulated as a liquid or ground-up into a powder from plants. The manufacturing process for liquid rotenone involves extraction with trichloroethylene (TCE) and toluene, followed by vacuum distillation to remove these solvents (USEPA, 2005). In addition, the liquid formulations may contain petroleum hydrocarbons as solvents and emulsifiers to disperse rotenone in water (primarily naphthalene, methylnaphthalenes, TCE and xylenes) (WDFW, 2002). The proportion of these carriers varies substantially by formulation, and formulations with synergists generally contain far less petroleum based carrier products. The potential impacts to ecological and human receptors associated with the adjuvants and carriers in the formulations proposed for use are discussed in Section 3.6.

Rotenone is the active ingredient in the commercially available piscicides Chem-Fish®, Cuberol®, Fish Nox®, Noxfire®, Nusyn-Noxfish®, Noxfish®, powder (Cube Powder Fish Toxicant®), and CFT Legumine®. Such formulations of rotenone include crystalline

preparations (approximately 95% pure), emulsified solutions (approximately 50% pure), and dusts (approximately 0.75-5% pure) (Extoxnet, 1996). This risk assessment considers the potential hazards and risks from the use of the CFT Legumine® and Noxfish® formulations, as only these formulations have been proposed for use in Lake Davis. Risks from the use of Cube Powder Fish Toxicant® as proposed under Alternative A are considered qualitatively, based on the inherent hazards of this source of rotenone as well.

J.3.2 Mechanism of Action of Rotenone to Fish

It was historically thought that rotenone suppressed oxygen uptake across the gills, eventually leading to death by suffocation (Schnick 1974). Recent studies, however, demonstrated that this is not the case, and rotenone has actually been shown to increase blood-oxygen concentrations in some fish species (Fajt & Grizzle 1998). Rotenone interrupts aerobic cellular respiration by blocking electron transport in mitochondria through the inhibition of the enzyme NADH ubiquitone reductase (Singer & Ramsay, 1994; Fukami et al., 1969; Lindahl & Oberg 1960) which prevents the availability of oxygen for cellular respiration. In other words, rotenone inhibits a biochemical process at the cellular level making it impossible for fish to use the oxygen absorbed in the blood and needed for the release of energy during respiration (Finlayson et al, 2000). In effect, rotenone causes death due to tissue anoxia with oxygen uptake blocked at the cellular level and not at the water/blood interface at the gills (Ling 2003). The lack of cellular oxygen availability initiates anaerobic respiration in turn leading to increased lactic acid concentrations resulting in a drop in blood pH level (Fajt & Grizzle 1998).

Rotenone is highly toxic to fish (Extoxnet 1996; WHO 1970) making it ideal for the control of invasive or unwanted fish species. In the aquatic environment, rotenone is readily transmitted across the permeable membranes of the gills. Gills are highly evolved respiratory structures that are able to maximize the uptake of O_2 and excretion of CO_2 because of the large surface area, thin lamellar membrane and efficient countercurrent exchange mechanism (Moyle & Cech, 1988). Fish are able to supplement this efficiency further by actively ventilating water across the gills by controlled branchial pumping. These features make fish highly susceptible to rotenone poisoning, even at very low concentrations. Variation in sensitivity to rotenone among fish species certainly exists, however, with rotenone tolerance generally varying inversely with oxygen requirements, as would be expected for a respiratory poison (Engstrom-Heg et al., 1978).

J.3.2.1 Bioconcentration, Bioaccumulation and Metabolism

Persistence of chemicals in biological tissues is commonly characterized through bioconcentration or bioaccumulation. Bioconcentration of a chemical can occur in an organism when it accumulates chemicals in its tissues following direct exposure, at a concentration greater than that found in the exposure media (e.g., water, air). If the organism is then consumed (i.e., predated upon) by another organism resulting in a higher concentration of the chemical in the predator, then the chemical is considered to bioaccumulate. Ney (1998) explains that bioaccumulation of organic chemicals in animals is a function of a chemical's ability to become soluble with fat. Fat-soluble (hydrophobic, non-

polar) chemicals are more prone to bioaccumulate in fatty tissues of animals because they are less prone to be metabolized by animals and will not, or will only slowly, dissipate or depurate when the animal is no longer exposed to the chemical. Chemicals that are insoluble in lipid exhibit polarity and are readily metabolized, dissipated and depurated in the animals.

Rotenone appears to bioconcentrate in aquatic organisms at acutely toxic concentrations but is detoxified and eliminated relatively fast when exposure concentrations do not lead to acute mortality. Rach & Gingerlich (1986) examined concentrations of rotenone and rate of breakdown in tissues in common carp *(Cyprinus carpio*), bluegill (*Lepomis macrochirus*), and yellow perch (*Perca flavescens*) following treatment until death. Common carp (*Cyprinus carpio*) exhibited the greatest tolerance to rotenone and contained the highest concentration with approximately 20 times that of the ambient water; bluegills were next with 8 times more rotenone found in their body; and yellow perch contained just 4 times the ambient rotenone concentration. These bioconcentration factors (BCFs) would be considered moderate, to low, relative to other organic compounds, which can exhibit BCFs orders of magnitude greater than rotenone.

Rach and Gingerlich (1986) also found that rotenone was quickly eliminated in carp with rotenoid metabolites accumulating in the bile. This confirmed results reported earlier by Fukami et al. (1969), who examined the detoxification of radio labelled rotenone by liver enzymes in carp. They found that rotenone was rapidly detoxified to a variety of hydroxylated rotenoids and more water soluble products with toxicities at least one to two orders of magnitude less than the parent rotenone. Thus the most likely route of detoxification and elimination is biliary excretion from the liver in the form of excretable metabolites.

Rotenone does not appear to bioconcentrate in the body with prolonged exposure at sublethal doses. Rotenone is rapidly detoxified by the mixed function oxidase (MFO) system of the liver enzymes which is responsible for rotenone breakdown. Fish that do not receive a fatal dose will recover relatively quickly with no further increase in toxicity as shown in 30-day flow through exposures performed by Marking and Bills (1976).

Absorption of rotenone in the stomach and intestines in mammals is relatively slow and incomplete. If absorbed, rotenone is metabolized rather effectively by the liver to produce less toxic excretable metabolites as shown by Ray (1991) in laboratory mammals. Approximately 20 percent of the oral dose (and probably most of the absorbed dose) is excreted within 24 hours as water soluble products with the remainder as hydroxylated rotenoids (Fukami et al. 1969). Large oral doses (200 mg/kg in pigeons and 10 mg/kg in dogs) usually stimulate vomiting in animals (Haag 1931 *as cited in* Ling 2003). Based on a review of results from these papers and others, Ling (2003) concluded that rotenone is not easily absorbed in higher animals and does not accumulate in the body. These results would also indicate that rotenone is not anticipated to bioaccumulate in increasing concentrations through food web consumption of exposed animals.

J.3.3 Environmental Fate and Chemistry

J.3.3.1 Physical Chemistry

Rotenone is a naturally occurring compound with empirical formula $C_{23}H_{22}O_6$ (Figure J-4) and a molecular weight of 394.43 (Extoxnet, 1996; FAO, 1970). It is derived from the roots of tropical plants (*Derris* spp., *Lonchocarpus* spp., and *Tephrosia* spp.) found in S. America,

Australia and parts of Southern Asia (USEPA, 2002). Rotenone is very soluble in a number of organic solvents such as alcohol and acetone but is only slightly soluble in water: 0.2mg/L at 20°C, 15mg/L at 100°C (Extoxnet, 1996).

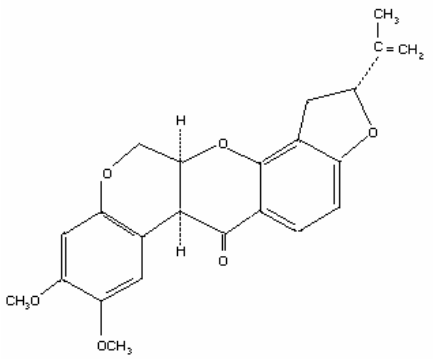


Figure J-4 Chemical Structure of Rotenone

J.3.3.2 Environmental Transport and Degradation of Rotenone

In mild temperatures, rotenone dissipates rapidly in both soil and water with a half-life between 1 and 3 days. It has a high tendency to adhere to soil particles and is unlikely to leach from soils; therefore, it is not likely to be a groundwater pollutant (Finlayson et al., 2001; Extoxnet, 1996). Rotenone is considered as a "highly active but short-lived photosensitizer" (Extoxnet 1996). This means that any organism consuming rotenone and unable to metabolize it, will become highly sensitive to the sun for a short period of time.

Wildlife consumption of rotenone-killed fish can be thought of as a means of environmental transport into other portions of the food web via the potential for accumulating body burden. In a thorough review of the literature, we have not identified empirical evidence to suggest that birds or mammals have died or become ill after eating fish killed by rotenone treatment, or by drinking treated waters. As previously discussed, the ability of birds and mammals to effectively neutralize rotenone in the gut by enzymatic action is largely thought to prevent bioaccumulation and adverse reactions from dietary and drinking water exposure. These physiological adaptations, coupled with the minute concentrations of rotenone generally found in dead fish will limit the extent that rotenone could be appreciably translocated through this pathway (i.e., via the body burden in fish) to other ecosystems.

Rotenone is very sensitive to light and temperature, and degrades rapidly in the presence of sunlight and warm temperatures (Extoxnet 1996). Rotenone persistence in natural bodies of water may vary from a few days to several weeks depending on the season (Ling, 2003;

Finlayson et al. 2001). Water temperature, light intensity, depth, dissolved oxygen concentrations, pH, turbidity, aquatic vegetation, and the presence of a thermocline may all affect the persistence and efficacy of rotenone.

Finlayson et al. (2001) conducted laboratory tests to record the degradation of rotenone in water at 4°C in the absence of light (Table J-2). They found that after six days, four out of six samples showed significant differences in rotenone concentration. Water with higher alkalinity (>170 mg/L $CaCO_3$) and pH (>9.0) had higher degradation rates of rotenone (-24% and -25%) than water with lower alkalinity (40mg/L CaCO3) and pH (7.7) (no change to –16%). As demonstrated in Table J-2, it would appear that the combination of high alkalinity and high pH are not required to accelerate degradation, as there was essentially no degradation recorded when just the pH was elevated without a concomitant increase in alkalinity. However, there was no test condition where high alkalinity and low pH were paired in this study.

Table J-2. Mean Rotenone Concentrations (μg/L) Before and After Six Days Storage at 4°C in the Absence of Light

Alkalinity (mg/L $CaCO_3$)	pH	Rotenone Before	Rotenone After	Percent change
40	7.8	91	93	+2
180	9.2	68	52	-24*
40	7.7	31.6	28.2	-11*
40	7.7	47.8	40	-16*
40	9.3	238	238	0
172	9.6	14	10.5	-25*

*Significant changes (p>0.05) using the Kruskal-Wallis test. (Source: Finlayson et al. 2001).

Gilderhus et al. (1986) conducted a study to determine the persistence of rotenone in the aquatic environment with respect to temperature (Table J-3). They concluded that rotenone degrades much quicker in warmer water—it degraded nearly 10 times faster at 23°C than at 1°C. They also discovered that the cold water treatment of 100 ppb remained toxic to rainbow trout 14 days after the initial rotenone treatment, even though the concentration measured was only 6 ppb. Similar findings were reported by Finlayson et al. (2001) after measuring the half-life of rotenone in several California reservoirs: Kaweah Reservoir (20-22°C), Frenchman Lake (10-22°C) and Lake Davis (5-12°C) had rotenone half-life values of 1.7, 3.5 and 7.7 days respectively (Table J-3).

Table J-3. Persistence of Rotenone in Ponds at Two Different Temperatures

Water Temperature	Initial Treatment: Rotenone Concentration	Time to decay to 0.02 mg/L	Half-Life of Rotenone
1°C	0.10 mg/L	11 days	83.9 hours, (3.5 days)
23°C	0.15 mg/L	48 hours (2 days)	13.9 hours, (0.5 days)

Source: Gilderhus et al., 1986
Note: Rotenone concentrations were analyzed by high performance liquid chromatography [HPLC]

Dawson et al. (1991) conducted a similar experiment in 1986. In addition to the effects of temperature on rotenone persistence they also examined the effects of sediment adsorption. The persistence of rotenone was compared between two ponds: one lined with cement, the other with an earthen-bottom. Studies were conducted during the spring, summer and fall to test difference in water temperature (Table J-4). Similar to the results of Gilderhus et al. (1986), the rate of rotenone degradation was positively correlated with increasing water temperature. In addition, for every temperature tested, rotenone disappeared two to three times quicker in the earthen pond versus the concrete lined pond. This finding supports the claim that rotenone has a high tendency to adhere to particles, in this case soil. However, while high initial sorption to the sediments was to be expected, rotenone did not apparently persist in the substrate and decreased to below limits of detection within 3 days of treatment, with water temperatures that ranged from 15 to 22°C. Dawson et al. (1991) also discovered that filtered water samples contained significantly less rotenone than the unfiltered samples. This result would imply that rotenone is readily adsorbed by suspended particles in the water column and not just the substrate.

Table J-4. Effects of Temperature and Sediment Adsorption on the Half Life (in Days) of Rotenone

Pond Substrate	Half Life of Rotenone (days)		
	Spring (8°C)	Summer (22°C)	Fall (15°C)
Concrete	3.7	1.3	5.2
Earthen	1.8	0.7	1.8

Source: Dawson et al., 1991

Rotenone aging studies conducted under laboratory conditions by Marking & Bills (1976) have highlighted the chemical's much shorter persistence when subjected to natural conditions. Half-lives for laboratory-aged solutions of rotenone in soft water were 13 days at 17°C and 22 days at 12°C, much longer than those found by Dawson et al. (1991) and Gilderhus et al. (1986) in field experiments. Furthermore, the toxicity of rotenone solutions declines in parallel with its chemical decay indicating that the breakdown products are comparatively non-toxic (Marking & Bills, 1976). Cheng et al. (1972) used photo degradation to identify the breakdown products of rotenone. They identified 20 separate products, most of which were rotenoids, only one of which (6aβ, 12αβ-rotenolone) is considered toxic (Cheng et al., 1972).

Recent field studies in California by Finlayson et al. (2001) lend support to previous conclusions about rotenone's rapid breakdown under natural conditions. They found that the estimated half-life of rotenone ranged between 0.58 and 7.7 days (mean of 2.3 days) depending on the water-body. Rotenone half-life values measured in four reservoir systems seemed to increase with increasing water depth, supporting the hypothesis that light is an important catalyst in rotenone degradation. Kaweah Reservoir, Success Reservoir, Lake Davis, and Frenchman Lake had half-life values measured at 1.7, 2.4, 7.7 and 3.5 days respectively (average depths of 8-12m) and Percolation Reservoir 12 and Meiss Lake had respective half-lives of 0.94 and 0.83 days (average depths of 0.8-1.0m) (Table J-5).

Table J-5. Rotenone Concentrations (μg/L) and Corresponding Half-Life ($t^1/_2$) Values In Lakes of Varying Depths

Location (Year)	Rotenone Concentrations (μg/L)				$t^1/_2$ (days)	Average Depth (m)
Kaweah Reservoir (1987)	76 (1)	55 (3)	43 (5)	<2 (12)	1.7	8-12
Bravo Reservoir (1987)	254 (1)	46 (2)	<2 (6)	---	0.65	---
Lonestar Pond (1987)	310 (1)	49 (2)	24 (6)	<2 (14)	1.8	---
Percolation Reservoir 5 (1987)	370 (1)	150 (3)	120 (8)	<2 (15)	1.7	---
Percolation Reservoir 12 (1987)	200 (1)	27 (3)	<2 (8)	---	0.94	0.8-1.0
Success Reservoir (1988)	122 (1)	39 (2)	22 (6)	<2 (30)	4.6	8-12
Meiss Lake (1988)	64 (0.13)	30 (1)	8.2 (3)	<2 (6.2)	0.96	0.8-1.0
Meiss Lake (1989)	47 (0.08)	41 (0.17)	30 (0.5)	18 (1)	0.96	0.8-1.0
Meiss Lake (1990)	11 (0.04)	5.9 (2.9)	3.8 (0.92)	<2 (1.9)	0.58	0.8-1.0
Frenchman Lake (1991)	90 (1)	39 (2)	28 (3)	6 (14)	3.5	8-12
Wolf Creek Lake (1992)	16 (8)	<2 (21)	<2 (28)	<2 (51)	2.9	---
Lake Davis (1997)	44 (1)	32 (3)	29 (7)	11 (21)	7.7	8-12

(Source: Finlayson Et Al. 2001)

Due to its low Henry's Law constant (1.1×10^{-13} atm-m^3/mol), rotenone is not expected to volatilize appreciably from surface water, and this estimation is supported by air quality modeling provided in Section 5 of the EIR/EIS, and summarized in the exposure assessment portion of this appendix (i.e., Section 4). The small amount of rotenone that may volatilize into the atmosphere will be readily degraded by reaction with photochemically-produced hydroxyl radicals. The half-life for this reaction in air is estimated to be 1.2 hours (NLM 2006).

J.3.4 Rotenone Toxicity to Ecological and Human Receptors

J.3.4.1 Toxicity to Fish

The efficacy of rotenone on various aquatic organisms has been examined in controlled aquatic toxicity tests. Such tests commonly aim to determine the LC_{50} value (the median water concentration of the active ingredient that kills 50 percent of the animals) over specified periods of time (e.g., 24 hours, 96 hours, etc.). Marking & Bills (1976) summarized such rotenone toxicity data for a variety of fish species (Table J-6). The tests used to

establish these values are conducted with laboratory quality water that lacks the colloid and sediment load typical of field settings. These organic loads consistently increase the amount of chemical required to elicit a toxic effect. Thus, lab values are conservative estimators of effects that could be seen in field settings.

Table J-6. Fish Toxicity of Noxfish®, Containing 5% Rotenone, in Standardized Laboratory Tests at 12°C

Species	Lethal Concentration of Noxfish®		Lethal Concentration of Rotenone (x 0.05)	
	LC_{50} 24h. (µg/L)	LC_{50} 96h. (µg/L)	LC_{50} 24h. (µg/L)	LC_{50} 96h. (µg/L)
Northern Pike	44.9	33.0	2.3	1.7
Atlantic salmon	35.0	21.5	1.8	1.1
Brook trout	47.0	44.3	2.4	2.2
Chinook salmon	49.0	36.9	2.5	1.9
Coho salmon	71.6	62.0	3.6	3.1
Lake trout	26.9	26.9	1.4	1.4
Rainbow trout	68.9	46.0	3.5	2.3
Goldfish	---	497.0	---	24.9
Common carp	84.0	50.0	4.2	2.5
Fathead minnow	400.0	142.0	20	7.1
Channel catfish	400.0	164.0	20	8.2
Black bullhead	665.0	389.0	33.3	19.5
Smallmouth bass	93.2	79.0	4.7	4.0
Largemouth bass	200.0	142.0	10	7.1
Green sunfish	218.0	141.0	10.9	7.1
Bluegill sunfish	149.0	141.0	7.5	7.1
Yellow perch	92.0	70.0	4.6	3.5
Longnose sucker	67.2	57.0	3.4	2.9
White sucker	71.9	68.0	3.6	3.4
Bowfin	57.5	30.0	2.9	1.5

Source: Marking & Bills, 1976

Rotenone applications of the commercial formulations between 1 and 3 mg formulation/L have generally proven sufficient to eliminate all fish in the treated water body (Ling, 2003). Such formulations result in active ingredient (a.i.,) concentrations of rotenone (i.e., rotenone) ranging from 50 to 150 µg/L (parts per billion). In such aquatic exposures, the water-borne chemical enters fish by simple diffusion across the gills. Marking & Bills (1976) recorded 24h LC_{50} rotenone concentrations of 1.4 µg/L to 33.3 µg/L, and 96h LC_{50} concentrations of a.i. ranging from 1.1 µg/L to 24.9 µg/L. Some of the most resistant species in field and lab applications have included black bullhead (*Ictalurus melas*), channel catfish (*I. punctatus*), and fathead minnow (*Pimephales promelas*) with 24-hour LC_{50} rotenone concentrations of 33.3 µg/L, 20 µg/L, and 20 µg/L, respectively. Salmonids (i.e., trout, salmon and char) tended

to be among the most sensitive species tested to the active ingredient with LC_{50} concentrations commonly less than 2.5 μg/L. Northern Pike (*Esox lucius*) demonstrate slightly less tolerance to rotenone than salmonids, with a 24-hour LC_{50} value of approximately 2.3 μg/L (Marking & Bills, 1976).

The considerable range in rotenone sensitivity among fish species has been exploited by fisheries managers to selectively remove populations of unwanted species in mixed-species communities (Bills et al., 1996). Reasons for such marked differences may be a result of differences in tissue distribution, rates of uptake, and rates of detoxification based on differences in the levels of liver enzymes responsible for rotenone breakdown and elimination, or supplemental means for oxygen uptake from air. Another possible explanation is that certain species are biochemically more successful in using alternative pathways to generate ATP (Rach & Gingerich, 1986) and therefore still able to function at some concentrations of rotenone that would otherwise kill other fish species.

Omnivorous fish species generally demonstrate higher tolerance levels to rotenone than strict carnivores. One explanation promoted for this elevated tolerance is that bottom-feeding omnivorous fish tend to have much greater concentrations of the mixed function oxidase (MFO) enzymes responsible for metabolizing rotenone than species with strictly carnivorous diets (Moyle & Cech, 1988). The MFO class of enzymes metabolize foreign compounds like rotenone, and accelerate their elimination, thus increasing the tolerance of such species with high rates of MFO induction to withstand otherwise lethal rotenone concentrations.

J.3.4.1.1 Effects of Physical and Behavioral Parameters on Rotenone Toxicity to Fish

Water-temperature and contact time are perhaps the two most important variables that modulate efficacy of rotenone treatments. Guilderhus (1972) found that the time required to achieve 100% mortality (LC_{100}) in various freshwater fish decreased approximately 2- to 3-fold for every five-degree increase in water temperature. Additionally, fish mortality will not occur if there is inadequate contact time between the chemical and the fish. Some fish species have been observed to demonstrate avoidance behaviors to rotenone, favoring areas with reduced concentrations, or areas that remain contaminant free (Hogue 1999). Therefore, in order to achieve complete elimination of the target species, it is essential to ensure an equal dispersion of rotenone throughout the water body.

Furthermore, fertilized fish eggs are less susceptible to rotenone poisoning than fishes themselves because their rate of toxicant uptake from the environment is much lower (Table J-7) (Ling 2003; Marking & Bills 1976). Programs aimed at eradicating a certain fish species should therefore take care to ensure the treatment falls either before the spawning season or after all eggs have hatched.

Water hardness and pH, and rotenone formulation can also modulate rotenone toxicity: Generally rotenone is reported to more effective when the natural body of water is somewhat acidic, with low hardness (i.e., soft water). However, Marking & Bills (1976) noted that the toxicity of rotenone to fish was not significantly affected by either water hardness or pH, but that its toxicity to newly fertilized fish eggs *decreased* with softer water (Table J-7). This

finding suggests that rotenone permeability through the egg chorion is diminished by softer water, a somewhat counterintuitive finding.

Table J-7. Toxicity of Rotenone in 12°C Water at Various Degrees of Hardness to Rainbow Trout and Rainbow Trout Eggs

	Median 96h LC_{50} (µg/L)			
	Very Soft Water	**Soft Water**	**Hard Water**	**Very Hard Water**
Rainbow trout (*O. mykiss*)	2.7	2.8	2.75	2.65
Newly fertilized *O.mykiss* eggs	280	221	160	125

Source: Marking & Bills, 1976

Following rotenone poisoning, fishes exhibit certain characteristic behaviors. In the induction stage of treatment reduced opercular ventilation coupled with erratic bursts of swimming is commonly observed. Surfacing and a 'gulping' behavior or skimming at the surface film may follow before a complete loss of equilibrium is experienced. Eventually, poisoned individuals sink to the bottom where they remain till death (Ling, 2003; Fajt & Grizzle, 1998; Rach & Gingerich, 1986).

J.3.4.2 Rotenone Toxicity to Non-target Aquatic Organisms

J.3.4.2.1 Aquatic Macroinvertebrates

With their gill-like tracheae, aquatic invertebrates are theoretically as susceptible to the toxic effects of rotenone as fish or amphibian larvae (Bradbury, 1986). After laboratory based tests, Chandler & Marking (1982), concluded that: apart from an Ostracod (*Cypridopsis sp.*), aquatic invertebrates are much more tolerant of rotenone than most fishes and amphibian larval stages. In their study the most resistant organisms exposed were a snail (*Helisoma sp.*) and the Asiatic clam (*Corbicula manilensis*) for which the LC_{50} 96h concentrations were 50 times greater than those Marking & Bills (1976) reported for the Black bullhead (*Ictalurus melas*), one of their most resistant fishes. Sanders & Cope (1968) also conducted lab tests examining the effect of rotenone to the nymph or naiad stage of a stonefly (*Pteronarcys californica*) They found that the LC_{50} 24h was 2,900 µg/L and the LC_{50} 96h was 380 µg/L. These values are greater by an order of magnitude to those found by Marking & Bills (1976) for the black bullhead (*Ictalurus melas*) indicating that aquatic invertebrates are much less sensitive to rotenone than fish. Larger, later instar naiads were less suceptible to given concentrations of toxin than were smaller, earlier instars of the same species (Sanders & Cope, 1968).

Field studies examining the effect of rotenone on aquatic macroinvertebrate communities have provided varied results. Whereas some workers noticed dramatic, long-term effects (Mangum & Madrigal, 1999; Binns, 1967), others observed rotenone has a negligible effect

on most aquatic macroinvertebrates (Demong, 2001; Melaas, 2001). Most researchers would agree, however, that the effects of rotenone are less pronounced and more variable to macroinvertebrates than the effects of the chemical on zooplankton. Like the range of sensitivities demonstrated by various fish species to rotenone, different species of aquatic macroinvertebrates also exhibit a range of tolerances (Mangum & Madrigal, 1999; Chandler & Marking, 1982; Engstrom-Heg et al., 1978) again perhaps based on their oxygen requirements (Table J-8).

Table J-8. Rotenone Toxicity Reported in Some Aquatic Invertebrates

Species Guild	Test Species	Test Endpoint	Lethal Concentration (mg/L)
Flatworm	*Catenula* sp.	LC_{50} 24h	5.1
	Planaria sp.	LC_{50} 24h	<0.5
Annelid worms	Leech	LC_{50} 48 h	<0.100
Copepod	*Cyclops* sp.	LC_{10}0 72h	<0.100
Branchiura	*Argulus* sp.	LC_{50} 24h	~0.025
Cladoceran	*Daphnia pulex*	LC_{50} 24h	0.027
	D. pulex	LC_{50} 24h	<0.025
	Diaptomus siciloides	LC_{50} 24h	<0.025
Conchostracan	*Estheria* sp.	LC_{50} 24h	~0.050
Freshwater prawn	*Palaemonetes kadiakensis*	LC_{50} 24h	5.15
Crayfish	*Cambarus immunis*	LC_{50} 72h	>0.500
Dragonfly naiad	Macromia sp.	LC_{50} 24h	4.7
Stonefly naiad	*Pteronarcys californica*	LC_{50} 24h	2.9
Backswimmer	*Notoncta* sp.	LC_{50} 24h	3.42
	Notonecta sp.	LC_{50} 24h	~0.100
Caddis fly larvae	*Hydropsychye* sp.	LC_{50} 96h	0.605
Whirligig	*Gyrinus* sp.	LC_{50} 24h	3.55
Water mite	Hydrachnidae	LC_{50} 96h	~0.050
Snail	*Physa pomilia*	LC_{50} 24h	6.35
	Oxytrema catenaria	LC_{50} 96h	1.75
	Lymnaea stagnalis	LC_{50} 96h	>1.0
Bivalve Mollusc	*Dreissena polymorpha*	LC_{50} 48h	0.219
	Obliquaria reflexa	LC_{50} 48h	>1.0
	Elliptio buckleyi	LC_{50} 96h	2.95
	Elliptio complanata	LC_{50} 96h	2
	Corbicula manilensis	LC_{50} 96 h	7.5
Ostracod	*Cypridopsis* sp.	LC_{50} 24h	0.490

Note: as summarized by Ling 2003, from a variety of sources

Invertebrates in the orders Ephemeroptera (Mayflies), Plecoptera (Stoneflies), and some members of Trichoptera (Caddisflies) are highly sensitive and have been completely eliminated by rotenone treatments in the past (Mangum & Madrigal, 1999). Areas lacking these 'sensitive' genera to begin with may therefore demonstrate a less pronounced effect of rotenone on the macroinvertebrate communities. Also, these sensitive species tend to be highly mobile and short life cycles, and may thus have the ability to repopulate depleted areas rapidly through dispersal and oviposition (Engstrom-Heg et al., 1978). Certain escape behaviors such as burrowing into benthos, associating with aquatic vegetation or the ability to trap air bubbles with appendages may reduce rotenone exposure to many benthic invertebrates. Also, chemical and physical features of the treated ecosystem will influence the toxicity of rotenone to the resident macroinvertebrate fauna as noted by Melass et al. (2001) with freshwater shrimp. Of note, many studies have shown rapid population explosions of invertebrates following initial reductions in their biomass from rotenone treatment (Neves 1975, Cook and Moore 1969).

J.3.4.2.2 Plankton

Changes in the abundance and/or structure of the plankton community, by the use of chemicals like rotenone can have marked effects on subsequent fish populations that depend on plankton either directly or indirectly for nutrition. From 1954-55 Hoffman & Olive (1961) conducted an experiment to document the effect of rotenone on the zooplankton community in a Colorado reservoir. They observed a complete kill of protozoans and Entomostracans and a major reduction in the Rotifer population following the treatment. Their finding agreed with previous research (Hooper, 1948; Brown & Ball, 1943; Hamilton, 1941) and more recent findings have demonstrated that rotenone is indeed highly toxic to zooplankton communities (Melaas et al., 2001; Beal & Anderson, 1993; Neves, 1975; Anderson, 1970; Kiser et al, 1963), especially in acidic conditions (Kiser et al. 1963). Unlike many benthic invertebrates, which may escape the immediate effects of rotenone by burrowing into sediment, zooplankton are exposed to rotenone for the full duration of its activity in the water column. However, some populations may recover from resistant life-stages and or eggs (Kiser et al. 1963). A full recovery of the zooplankton community may take longer however. Beal & Anderson (1993) demonstrated that some populations make take up to 8 months to recover following rotenone treatment, while Anderson (1970) noted a 3-year recovery period in two mountain lakes. Therefore, if rotenone is used with a future restocking program in which naturally produced food items are depended on, time must be given for the zooplankton communities to re-establish themselves before fish are re-introduced into the lake.

J.3.4.3 Toxicity to Terrestrial Wildlife Receptors

As rotenone is commercially used as both an insecticide and a piscicide, it follows that it can be toxic to both aquatic and terrestrial species—again depending on the dose, method of administration, duration of exposure, and sensitivity of the species and life stage. Table J-9 details chemical toxicity guidelines established by the USEPA which are often used as guidance to gauge the toxicity of rotenone and other chemicals to mammals and birds. It

provides a good backdrop by which to consider results from past studies. Two hazard categories are listed in the table, the acute oral or dermal LD_{50} and the acute inhalation LC_{50}. The LD_{50} is the statistical derivation of a dietary or drinking water dose, predicted to cause 50% mortality in the given population being tested. The LC_{50} is a similar number, based on the concentration of a compound in air or water.

Table J-9. Toxicity of Rotenone to Various Amphibians in Lakes

Species	Stage	Temp °C	24 hours LC_{50} (µg/L)	96 hours LC_{50} (µg/L)	Original Reference
N. Leopard frog (*Rana pipiens*)	Juvenile/ Adult	___	10	___	Haag, 1931
	Tadpole	___	5	___	Hamiliton, 1941
	Adult	12	240	240	Farringer, 1972
	Adult	12	1200	290	Farringer, 1972
	Adult	12	1460	920	Farringer, 1972
	Adult	12	1580	640	Farringer, 1972
Tiger salamander (*Ambystoma tigrinum*)	Larvae	___	5	___	Hamilton, 1941
S. Leopard frog (*Rana sphenocephala*)	Tadpole	15-17	30	25	Chandler & Marking, 1982

J.3.4.3.1 Rotenone Toxicity to Mammals

Mammalian acute oral toxicity LD_{50} values for rotenone range from 39.5 mg/kg for female rats to 1,500 mg/kg for rabbits. For most lab mammals, rotenone is much more toxic when introduced intravenously or inhaled rather than taken orally. For example, the average oral LD_{50} for rats is 60 mg/kg compared with just 0.2 mg/kg for rotenone introduced directly into the bloodstream. Efficient breakdown of rotenone by the liver, oxidation of rotenone in the gut, and slow absorption in the stomach and intestines account for this significant difference in toxicity (Narongchai et al. 2005; Ling 2003). This explanation may also account for the significant difference in rotenone sensitivity between mammals and fishes, and not from a difference in the primary site of action between fishes and mammals (Fukami et al., 1969). Indeed, The USEPA considers rotenone safe to use in the presence of cattle (USEPA 1981).

J.3.4.3.2 Rotenone Toxicity to Birds

Rotenone has a very low toxicity to wildfowl, and birds are extremely unlikely to be affected by 'normal' usage in fisheries management practices (Ling, 2003). Avian acute toxicity LD_{50} values range from 130mg/kg for the nestling English song sparrow (Cutcomp 1943) to 2200mg/kg for an adult mallard duck (USEPA 1988). In general, young birds are about 10 times more sensitive to rotenone poisoning (DFG 1994) and, like mammals, birds have a much-reduced tolerance to rotenone when it is introduced intravenously. During recent rotenone treatments in California, fish-eating birds and mammals were observed foraging on

dying and recently deceased fishes for several days following treatment. There were no reported sightings of dead birds or mammals over the following days and weeks (DFG 1994).

Ling (2003) also examined rotenone poisoning and sublethal toxicity in birds as a result of consuming fish or even fish management baits. Ling concluded that "rotenone is slightly toxic to wildfowl, and birds are extremely unlikely to be affected by normal fisheries management programmes." For example, baits used to kill carp for management purposes have around 0.01 g of rotenone each. Ling calculated that a duck would need to consume approximately 200 baits to receive a fatal dose. It is very unlikely that birds would consume baits but they could consume fish killed by rotenone. The concentration of rotenone in poisoned fish is usually 25,000 times lower than that found in baits.

J.3.4.3.3 Rotenone Toxicity to Terrestrial Insects

Rotenone is extremely toxic to many species of insects in many different insect orders (caterpillars, beetles, flies, etc.) hence its wide popularity as an insecticide. However, the compound is considered to be non-toxic to bees unless used in combination with pyrethrum (Extoxnet 1996). Given that the use of rotenone by fisheries management practices will be restricted to the aquatic environment, only aquatic insects or aquatic stages of terrestrial insects could be significantly affected.

J.3.4.3.4 Rotenone Toxicity to Amphibians

Rotenone is toxic to amphibians, but generally less toxic than to fish. Rotenone may have be absorbed into both skin and respiratory membranes, but skin may prevent more of a barrier due to a greater distance for the chemical to diffuse across (Fontenot et al., 1994), and a smaller surface area relative to gill structure. Indeed, Fontenot et al. (1994) reported that amphibian larvae with gills are most sensitive to rotenone. In early 1974, African clawed frogs (*Xenopus laevis*) were discovered in some ponds located in the Santa Clara River drainage. An eradication program using rotenone to extirpate the exotic frogs was undertaken in the spring of 1974. Results indicated that all *X. laevis* tadpoles had been killed but adults were unaffected and thus able to reproduce again later that spring (McCoid & Bettoli, 1996).

In standard laboratory 24-hour and 96-hour aquatic rotenone toxicity tests, the LC_{50} values for tadpoles and larval amphibians have ranged between 5 μg/L and 580 μg/L (24-hour tests and 25 μg/L to 500 μg/L in 96-hour tests (Fontenot et al. 1994, Chandler 1982). The adult Northern Leopard Frog demonstrated a much greater resistance with LC_{50} concentrations ranging from 240 μg/L and 1,580 μg/L (24 hours) and 240 μg/L and 920 μg/L (96 hours) (Table J-9). This highlights the fact that tadpoles and other larval forms of amphibians that utilize gills for respiration are just as sensitive to rotenone as fishes while adult forms, no longer having to utilize gills, have a much lower susceptibility to rotenone. Larval amphibians appear to have resistance roughly equivalent to the most tolerant fish species.

J.3.4.3.5 Rotenone Toxicity to Reptiles

Studies of rotenone toxicity to reptiles are particularly lacking (Fontenot et al. 1994). Carr (1952) and Dundee & Rossman (1989) suggested that soft-shelled turtles (*Apalone* spp.) may

be affected by rotenone applications in fisheries, although neither provided data to support their statements. The adult Green Anole (*Anolis carolinensis*) is the only reptile species for which pre-registration testing of chemicals, including rotenone compounds, for acute lethal toxicity has been considered (Fontenot et al. 1994). Aquatic turtle species with specialized respiratory mechanisms such as buccopharyngeal respiration (*Apalone spinifera* and *Kinosternon minor*), or modified skin & cloaca to enhance respiration (*Trachemys scripta* and *K. odoratum*) may be more susceptible to the effects of rotenone than other more terrestrial species. Turtle species in the family kinosternidae generally possess these special respiratory systems (Fontenot et al. 1994).

A fish population study using rotenone on Lake Conroe (Montgomery County, Texas) conducted between 1980 and 1986 indicated that aquatic turtles (*K. subrubrum*) were indeed susceptible to rotenone poisoning. At least 60 dead or dying individuals were observed around the periphery of the lake 24–48 hours after treatment (McCoid & Bettoli, 1996). This is thought to be a very conservative figure however as K. subrubrum tends to sink when dead (McCoid & Bettoli, 1996). Freshwater aquatic snakes do not utilize aquatic respiration and absorption of rotenone through the thick skin is considered very unlikely (Fontenot et al., 1994). One study (Haque, 1971) however, reported the death of an aquatic snake in a pond 48 hours after treating with rotenone, but noted a second healthy-looking snake swimming in the same pond at the time. The mechanism of action of uptake and toxicity of rotenone to reptiles requires further study.

J.3.4.4 Summary of Toxicity Reference Values (TRVs) Used for Ecological Risk Assessment

Table J-10 summarizes the range of acute and chronic toxicity reference values (TRVs) identified for rotenone for terrestrial species. These TRVs will be compared against estimated exposure doses calculated from species-specific parameters outlined the Exposure Assessment (Section 4.1), and used subsequently to calculate hazard quotients for risk characterization (Section 5.1). Based on EPA hazard classifications (Table J-11), and the toxic effect doses identified in Table J-10, rotenone would qualify as moderately toxic to highly toxic to mammals, and moderately toxic to practically non-toxic to birds—depending on the species and life stage considered.

Table J-12 and Table J-13 summarize the hazard quotient calculations and the levels of concern presumed for characterizing risk associated with these hazard quotients, for non-target aquatic and terrestrial animals, respectively.

Table J-10. Acute Toxicity of Rotenone to Selected Mammalian and Avifauna

Animal Group	Toxicology Test	Median Lethal Concentration	Reference(s)
Mammals			
Human	Acute LD_{50} oral	300-500 mg/kg-body wt (Estimated)	USEPA, 1988
Rat	Acute LD_{50} oral	39.5 mg/kg (female)	USEPA, 1988

Table J-10. Acute Toxicity of Rotenone to Selected Mammalian and Avifauna

Animal Group	Toxicology Test	Median Lethal Concentration	Reference(s)
	Acute LD_{50} oral	102 mg/kg (male)	USEPA, 1988
	Chronic NOAEL TRV	0.4 mg/kg-bw/day	USFWS, 1983
	Chronic LOAEL TRV	2 mg/kg-bw/day	USFWS, 1983
Mouse	Acute LD_{50} oral	350 mg/kg	Kidd & James, 1991; USEPA, 1988
Guinea pig	Acute LD_{50} oral	12-200 mg/kg	USEPA, 1988
Rabbit	Acute LD_{50} oral	600-2000 mg/kg	USEPA, 1988
	Acute LD_{50} oral	~1500 mg/kg	Unknown reference
Dog	Chronic NOAEL TRV	0.4 mg/kg-bw/day	USFWS, 1980
	Chronic LOAEL TRV	2 mg/kg-bw/day	USFWS, 1980
Birds			
English song sparrow (nestling)	Acute LD_{50} oral	130 mg/kg	Cutcomp, 1943 (in DFG, 1994)
American robin (nestling)	Acute LD_{50} oral	200 mg/kg	Cutcomp, 1943 (in DFG, 1994)
Quail	Acute LD_{50} oral	1882 mg/kg	Unknown reference
Mallard duck	Acute LD_{50} oral	2200 mg/kg	USEPA, 1988
	Acute LD_{50} oral	> 2000 mg/kg	Extoxnet, 1996
Pheasant	Acute LD_{50} oral	1680 mg/kg	USEPA, 1988
	Acute LD_{50} oral	>1680 mg/kg	Extoxnet, 1996

Table J-11. Chemical Hazard Classifications for Wildlife Risk

Hazard Category	Mammals Acute Oral or Dermal LD50 (mg/kg)	Mammals Acute Inhalation LC_{50} (ppm)	Avian Acute Oral or Dermal LD_{50} (mg/kg)	Avian Acute Onhalation LC_{50} (ppm)
Very highly toxic	<10	<50	<10	<50
Highly toxic	10-50	51-500	10-50	51-500
Moderately toxic	51-500	501-1000	51-500	501-1000
Slightly toxic	501-2000	1001-5000	501-2000	1001-5000
Practically non-toxic	>2000	>5000	>2000	>5000

USEPA 1995

Table J-12. Risk Presumptions for Non-Target Fish and Aquatic Invertebrates Exposed to Rotenone Formulation Constituents from Lake Davis Treatment for Northern Pike Eradication

Toxicity Endpoint	Hazard Quotient (HQ) Calculation	Level of Concern (LOL) with Hazard Quotient
Acute Exposure	EPC[1]/LC50[2] *or* EC50[3]	0.5
Acute Restricted Use Exposure	EPC/LC50 or EC50	0.1
Acute Endangered Species Exposure	EPC/LC50 or EC50	0.05
Chronic Exposure	EEC/NOAEC[4]	1

Source:USEPA 1995

1: Exposure point concentration in primary media of exposure (*aka* environmental exposure concentration)

2: Median lethal concentration of chemical that kills 50% of the test organisms

3: Median effective concentration of chemical that elicits measurement of effect in 50% of the test organisms

4: No observable adverse effect concentration

Table J-13. Risk Presumptions for Non-Target Terrestrial Animals Exposed to Rotenone Formulation Constituents from Lake Davis Treatment for Northern Pike Eradication

Toxicity Endpoint	Hazard Quotient (HQ) Calculation	Level of Concern (LOL) with Hazard Quotient
Acute Exposure	EPC[1]/LC50[2] *or* EC50[3]	0.5
Acute Restricted Use Exposure	EPC/LC50 or EC50	0.2
Acute Endangered Species Exposure	EPC/LC50 or EC50	0.1
Chronic Exposure	EEC/NOAEC[4]	1

Source: USEPA 2005

1: Exposure point concentration in primary media of exposure (*aka* environmental exposure concentration)

2: Median lethal concentration of chemical that kills 50% of the test organisms

3: Median effective concentration of chemical that elicits measurement of effect in 50% of the test organisms

4: No observable adverse effect concentration

J.3.5 Human Toxicity from Rotenone

Rotenone products formulated for use in fisheries management have been classified by the USEPA as Category 1 materials, which are in the "extremely toxic" range for acute (short-term) toxicity to humans. This classification is provided for exposure concerns for workers who formulate the pesticide mixtures that are then applied to the environment. Concentrations of the active and inactive ingredients are lower once the mixture has been applied to the environment, especially when it is applied to a large water body such as Lake Davis. Inhalation, dermal, and ocular exposures are the three most common routes of

exposure for people who apply the chemical mixtures according the regulatory requirements. These types of exposures are significantly mitigated by the use of proper handling procedures and protective equipment such as air-purifying respirators, protective clothing (coveralls, gloves), and eye protection (splash goggles or face shields) (Finlayson, et, al, 2000).

Anecdotal evidence suggests human poisoning has occurred with some regularity following the deliberate or accidental ingestion of roots containing rotenone in Papua New Guinea (Wood et al. 2005) and Thailand (Narongchai et al. 2005). Individuals who were known to have intentionally ingested the plant roots were reported to suffer from profound vomiting, dilated pupils and feeble pulse before death. Autopsies in fatal cases showed acute congestive heart failure (Wood et al, 2005).

Until recently there had been no reports of poisoning with commercially available rotenone. De Wilde et al. (1986) reported the death of a 3½ year-old girl in Belgium caused by respiratory arrest after apparently swallowing a mouthful of an insecticide product called "Galicide." Galicide is a French-manufactured insecticide containing 6.1% rotenone. An autopsy of the child discovered rotenone at levels of 2 to 4 parts per million (ppm) or 20-40 mg/kg in the blood, liver, and kidney. The child was reported to have suffered from vomiting, severe metabolic acidosis, drowsiness, coma and respiratory depression leading to respiratory arrest prior to death—symptoms similar to those reported in Papua New Guinea. Postmortem studies showed anoxic damage to the brain, lungs and heart, with an associated hemorrhaging of the lung, acute tubular necrosis and significant gastrointestinal irritation and hemorrhage (Wood et al, 2005). The authors reported that although values of 2 to 4 ppm seemed somewhat low, they considered it very likely that these amounts resulted in the victim's death. Wood et al. (2005) since reported on the death of a 47-year-old woman in the United Kingdom which resulted from consuming 200 ml from a bottle of 0.8% rotenone solution. Taking her weight and the dosage into consideration equates to a rotenone dose of 25 mg/kg. In both incidents the fatal dosage is considerably less than of those reported for other mammals in Table J-10. However, emulsive oils present in both rotenone formulations are likely to have enhanced the solutions' toxicity.

In humans the minimum lethal dose is not known, but death occurred in the 3½ year-old child who had ingested 40 mg/kg rotenone solution (Wood et al, 2005). This concentration is much higher than what would be present in the reservoir water for the effective piscicide dose. No reports were identified of human health effects following appropriate protocols for application of rotenone as a piscicide, thus, the understanding of potential human health effects must be extrapolated from controlled animal studies and case reports from accidental or intentional deaths associated with ingestion of high doses of rotenone.

J.3.5.1 Interpretation of Human Toxicity Based on Animal Studies

Due to the lack of data directly addressing human exposure to rotenone, the toxic effects of rotenone exposure on humans can best be understood from controlled acute animal studies. The toxicity of rotenone in animal studies has been demonstrated to be variable. The 50% lethal dose (LD_{50}; i.e. the median dose required to kill 50% of the animal populations studied) varied from 13 to 130 mg/kg in guinea pigs and from 25 to 132 mg/kg in rats to 1,500 mg/kg in rabbits (Wood et al, 2005). The differences observed in the fatal doses of

rotenone may reflect differences in the preparations that were used or ingested, in addition to species differences in toxicity (Wood et al, 2005). For example, higher doses are required to demonstrate toxicity for water-based preparations compared to fat-based preparations. This is consistent with the properties of rotenone, since is very poorly soluble in water. In addition, the route of administration of the dose was a key factor in determining the LD_{50}. If the dose was administered by subcutaneous, intravenous, or intraperitoneal routes, the LD_{50} was much lower than for oral doses. This likely reflects the rapid first pass metabolism of rotenone by the liver (Wood et al, 2005), a metabolic detoxification process that is avoided with the direct injection exposure pathways.

It has also been demonstrated in rat studies that rotenone appears to be more toxic to female rats (LD_{50} = 39.5 mg/kg) compared to male rats (LD_{50} = 102 mg/kg), based on an acute oral exposure (USEPA, 2005) For females, formulated end-product (LD_{50} = 130 mg/kg bw) was also roughly 3 times less toxic than technical grade (LD_{50} = 39.5 mg/kg bw). These data suggest that cube root extractable compounds do not contribute appreciably to the toxicity of rotenone (USEPA 2005). Formulated product toxicity testing data were available on three products used as applications to water for fishery resource management purposes. All of the formulations tested (varying from 2.6 to 8.08% rotenone) were less toxic than technical grade active ingredient on an acute oral exposure basis (USEPA 2005). In addition, similar to the technical grade rotenone, all of the formulated products tested were more toxic to female rats than to male rats by factors ranging from 1.6 to 5.0X (USEPA 2005)

Classical signs of acute toxicity following ingestion exposure of animals to rotenone include: initial respiratory stimulation, followed by significant respiratory depression and respiratory arrest (Wood et al, 2005). Death occurs in the first 30 minutes in roughly half of animals given substantial intraperitoneal doses of rotenone, and within 2 days in animals that orally ingested rotenone (Wood et al, 2005). Other effects observed in animal studies include vomiting, incoordination, convulsions, and muscular tremors with postmortem studies on the deceased animals demonstrating pulmonary congestion and gastrointestinal irritation (Wood et al, 2005).

Because rotenone is unstable in the environment, the focus of toxicity studies has been on acute effects rather than chronic effects. The limited chronic studies available on rotenone are chronic studies evaluating reproductive effects on animals. These studies indicate that the primary chronic toxic effect was adult and offspring decreased body weight (USEPA 2005).

J.3.5.2 Review of Parkinson's Related Potential Effects

Concern has been raised about studies showing a potential link between Parkinson's disease and rotenone. USEPA (2005) and the Washington Department of Fish and wildlife (WDFW) cited an Emory University study conducted in 2000 which reported finding a relationship between Parkinson's disease and rotenone exposure. According to USEPA and WDFW, the Emory University study demonstrated that rotenone produced Parkinson's like anatomical, neurochemical, and behavioral symptoms in laboratory rats when administered chronically and intravenously. In this study, 25 rats were continuously exposed for 5 weeks to 2 to 3 mg rotenone (dissolved in dimethyl sulfoxide [DMSO] and polyethylene glycol [PEG]) per kg body weight per day. The exposure was accomplished by injecting the mixture directly into

the right jugular vein of the rats using an osmotic pump. Twelve of the 25 rats developed lesions characteristic of Parkinson's disease. Structures similar to Lewy bodies (microscopic protein deposits) in the neurons of the *substantia nigra* in the brain (characteristic of Parkinson's disease) were produced in several of the rotenone-exposed rats. Dr. J. T. Greenamyre who directed the Emory study had indicated that because these effects were observed in rats that presumably the same can happen in people.

Dr. Joseph Borzelleca of the Virginia Commonwealth University Department of Pharmacology and Toxicology and an extensively published Pharmacologist/Toxicologist; researcher; journal editor; consultant to the World Health Organization and member of National Academy of Science Committee on Toxicology, critically reviewed the Emory University study to determine its relevance for humans (WDFS 2002).

Dr. Borzelleca indicated that a more relevant study for human exposure considerations was conducted by Marking in 1988, which involved administering rotenone in the diet to male and female rats for 2 years (lifetime for rats) at doses up to 75-mg/kg-body weight/day. The study results indicated no changes to the brains of the rats that had eaten rotenone daily for two years and, the rats did not develop any signs of Parkinson's disease during the course of the study. Dr. Borzelleca indicated that the Marking's study is relevant for human exposure because entry into the body was with food (simulates the human condition); in addition, the doses in the Marking study were about 30 times greater (2.5 versus 75 mg/kg-body weight/day) than the Emory study and the exposure was much longer (2 years versus 5 weeks) than in the Emory study (as cited in WDFW, 2002). Therefore, there is a high level of confidence that the Marking oral exposure study adequately reflects potential human exposure conditions.

Both the USEPA (2005) and WDFW (2002) emphasized that the Emory study is not relevant to assessing potential toxic effects to humans as a result of exposure to rotenone following its use as a piscicide. In the Emory study the manner that rotenone was administered to the laboratory rats is not an exposure condition that would occur in the environment. Not only was it administered by continuous jugular vein infusion but it was also mixed with DMSO and PEG. DMSO enhances tissue penetration of many chemicals. Direct injection is the fastest way to deliver chemicals to the body, as evidenced in intravenous application of medicines. Continuous intravenous injection, as done in the Emory University study, also leads to continuous high levels of the chemical in the bloodstream. The normal exposure to rotenone in humans from its use in fisheries management would be incidental ingestion, inhalation, or dermal contact through the skin (WDFW, 2002). Therefore, for these reasons, the method of exposure in the Emory University study cannot be used as a model for any form of rotenone exposure resulting from its use in fisheries management [Rotenone Stewardship Program 2001 as cited by WDFW (2002)].

Unlike the Emory study which involved direct injection of rotenone in the test species, exposure of humans to rotenone in the environment is extremely limited because rotenone is very unstable, is oxidized (neutralized) through enzymatic action in the gut of mammals and birds, is metabolized to water-soluble compounds in the body, and these compounds are excreted by the liver and kidney (WDFW, 2002). Because of the rapid metabolism and clearance in mammals and birds, it is not likely that rotenone could reach the site of action in

the substantia nigra in the brain where the dopamine is formed. Rotenone is toxic to fish because it is taken up rapidly across the gills and gets directly into the bloodstream, thus bypassing the gut. Rotenone is considered safe for the environment because it is not persistent and loses all its toxicity in a few days in lowland lakes (WDFW, 2002). In fact, it is significant that the Emory University investigators could not administer rotenone in any other manner except intravenously and get delivery of rotenone to the brain; otherwise, rotenone would have been neutralized in the gut and liver of the rats (Rotenone Stewardship Program, 2001 as cited in WDFW, 2002).

J.3.5.3 Summary of Rotenone Toxicity and Regulatory Screening Values for Human Health Risk Assessment

As explained in section 2.5.2, toxicity in humans is categorized by non-carcinogenic and carcinogenic responses. This current assessment evaluates the potential for both responses.

For evaluating chronic noncancer human health effects due to rotenone exposure, USEPA has developed an oral reference dose (RfDo) of 0.004 mg-rotenone/kg/day based on the results of chronic (long term) toxicity studies with rats (USEPA/IRIS 2006). This value was based on a reproductive study where rats were fed diets containing 0, 7.5, 37.5, or 75 ppm (0, 0.38, 1.88, or 3.8 mg/kg/day) rotenone through two generations. Litter sizes were reduced in the 75 ppm (3.8 mg/kg/day) dose group (highest dose tested) and pup weights were reduced in both generations during lactation for the 37.5 and 75 ppm dose groups. Body weights and body weight gains in adult rats were reduced during the two generations also. Based on these results, the lowest observable adverse effect level (LOAEL) for reproductive toxicity, a chronic effects measurement endpoint, is 37.5 ppm (i.e., 1.88 mg/kg/day ~2 mg/kg/day) and the no-observed adverse effects level (NOAEL) is 7.5 ppm (0.38 mg/kg/day, or ~0.4 mg/kg/day). An uncertainty factor of 100 was used to account for the inter- and intraspecies differences to arrive at the chronic RfDo of 0.004 mg/kg/day for humans (USEPA/IRIS 2006).

It should be noted that the conditions of this chronic two generation study are not representative of the conditions in the environment that will be present following the Lake Davis rotenone application. Degradation in the environment of the rotenone and other compounds in the formulations should reduce exposure time to acute (short term) or at worst sub-chronic conditions rather than chronic. Therefore, for noncancer risks, sub-chronic reference doses, which are relevant to exposure scenarios not likely to exceed a few weeks, will be used to estimate exposure for the media to which humans could be exposed—in this case, water, soil and air.

The National Academy of Science (NAS 1983 as cited in Finlayson, et al, 2000) has suggested a Suggested No-Adverse Response Level (SNARL) for rotenone in drinking water of 14 µg/L. The California Department of Health Services has suggested an Action Level (level of concern) for rotenone in drinking water of 4 µg/L. These proposed life-time, allowable levels for drinking water are based on applying a 1,000-fold safety factor to the chronic feeding study of Ellis et al. (1980 as cited in Finlayson, et al, 2000). For comparison, most rotenone treatments are done within the range of 25–250 µg/L, and rotenone generally

persists for no longer than a few weeks. In addition, rotenone treatments are only infrequently applied to any body of water.

Based on the most current toxicological information available from USEPA's Integrated Risk Information System (IRIS), USEPA Region 9 has established human health-based screening levels for rotenone in tap water, soil/sediment, and air and refers to these levels as preliminary remediation goals (PRGs). The tap water screening level for rotenone is 150 μg/L, for residential soil/sediment the value is 240 mg/kg, and for air 180 μg/m3 (USEPA 2004).

According to the USEPA (1988), rotenone has not been classified as a potential carcinogen. However, some of the other formulation constituents have carcinogen WoE classifications. A complete summary of the non-carcinogenic and carcinogenic human health based screening values for rotenone and other components in the CFT Legumine and Noxfish are consolidated in Table J-14. Additional discussion of the fate, transport, and toxicity of the other formulation constituents is provided in Section 3.6.

Table J-14. Noncancer Subchronic Reference Doses and Cancer-Based Slope Factors for Rotenone and the Principal Formulation Constituents

Component	Formulation		Subchronic Reference Dose (RfD)		Cancer Slope Factor (CSF)			
	CFT Legumine	NoxFish	Oral (mg/kg/day)	Inhalation (mg/kg/day)	Oral (mg/kg/day)[1]		Inhalation (mg/kg/day)[1]	
Rotenone	√	√	0.004 [a,b #]	0.004 [a,f #]		nc		nc
Butylbenzene, 1-	√	√	0.11 [b,c]	0.11 [b,c]		nc		nc
Butylbenzene, sec-	√		0.11 [b,c]	0.11 [b,c]		nc		nc
Isopropylbenzene		√	0.4 [d]	1.1 [a,l]		nc		nc
Isopropyltoluene, 4-	√	√	0.8 [a,b,e]	1.4 [a,e,l #]		nc		nc
Methylnaphthalene, 2-	√		0.004 [a,b #]	0.004 [a,b #]		nc		nc
Naphthalene	√	√	0.2 [a,b]	0.00086 [a,l #]	C	0.12°	C	0.12°
Propylbenzene, 1-		√	0.11 [b,c]	0.11 [b,c]		nc		nc
Toluene		√	0.8 [a,b]	1.4 [a,l #]		nc		nc
Trichloroethene		√	0.0003 [f,g #]	0.17 [m,n #]	X	0.013°	X	0.007°
Trimethylbenzene, 1,2,4-		√	0.5 [b,h]	0.0051 [h,l]		nc		nc
Trimethylbenzene, 1,3,5-	√	√	0.5 [h]	0.017 [h]		nc		nc
Xylene, 1,2-		√	0.2 [a,b,i #]	0.086 [a,i,l]		nc		nc
Xylene, 1,3- and/or 1,4-		√	0.2 [a,b,i #]	0.086 [a,i,l]		nc		nc
Diethylene glycol monoethyl ether	√		0.6 [h]	0.0086 [h]		nc		nc
Methyl-2-pyrrolidinone, 1-	√		0.043 [j #]	0.043 [j #]		nc		nc
Rotenolone	√	√	nd	nd		nc		nc
Potassium permanganate			0.0075 [k #]	nd		nc		nc

nc = not carcinogenic.
nd = not determined.
LD_{50} = dose lethal to 50% of a study population.
Carcinogenic classifications:
C = possible human carcinogen (limited evidence of carcinogenicity in animals with no human data)
X = wieght-of-evidence of human carcinogenic potential has been removed and is under review by EPA.

[#] Subchronic RfD is the same as chronic RfD.
[a] USEPA, IRIS, 2006.
[b] Based on chronic oral RfD and applicable uncertainty factors.
[c] USEPA, NCEA RA Issue Paper 99-010, 1999.
[d] USEPA, HEAST, 1997a.
[e] Toluene used as a surrogate.
[f] Chronic oral RfD.
[g] NCEA provisional value (USEPA-9, PRGs, 2004b).
[h] USEPA, PPRTVs, 2004a.
[i] RfD for mixed xylenes.
[j] Acute oral rat LD_{50} of 3,914 mg/kg (NLM, HSDB On-Line Database, 2006) / uncertainty factor of 100,000.
[k] Acute oral rat LD_{50} of 750 mg/kg (NLM, HSDB On-Line Database, 2006) / uncertainty factor of 100,000.
[l] Based on chronic inhalation RfD and applicable uncertainty factors.
[m] Chronic inhalation RfD.
[n] Cal/EPA, OEHHA Chronic RELs, 2000.
[o] Cal/EPA, OEHHA Toxicity Criteria Database, 2005.

J.3.6 Environmental Fate and Hazards from Carrier and Dispersant Ingredients in the Proposed Rotenone Formulations, and the Potassium Permanganate Neutralizing Agent

Chemical constituents in the commercial rotenone formulations proposed for Lake Davis may pose toxic risks to human populations or ecological receptors if the expected environmental concentrations (EEC) in exposure media or the estimated uptake by specific receptor populations (i.e., doses) exceed established toxicity thresholds and/or regulatory criteria. Thus, these constituents, along with rotenone constitute the 'chemicals of potential concern' (COPCs) for which additional hazards and risks to human and ecological receptors are examined in this technical appendix. This section examines what is known about the fate and hazards of other chemical constituents in the rotenone formulations. The environmental fate of potassium permanganate, the rotenone neutralizing compound proposed for use in Big Grizzly Creek to ensure rotenone toxicity is not conveyed downstream (see EIS, Section 2.3.4), is also considered here. Regulatory criteria do not exist for all constituents in all media that can be monitored (e.g.,, air, water, sediment). Further, there exist no TRVs for many ecologically relevant toxicity endpoints.

J.3.6.1 Physical and Chemical Properties of of Carrier and Dispersant Ingredients in Rotenone Formulations

While the CFT Legumine® and NoxFish® formulations considered for use in Lake Davis are reported by the manufacturer to contain the same concentration of rotenone (5%), the concentrations and types of dispersant and carrier compounds in the two formulations differ substantially. Table J-15 summarizes some of the physical and chemical characteristics of rotenone compared to the various inert ingredient and carrier compounds present in the two rotenone formulations. The physical and chemical characteristics of a compound determine its fate in the environment. The rate and manner of the breakdown of each chemical is dependent on its solubility, volatility, tendency to adsorb to soil or sediment particles, and other factors shown in this table. As demonstrated in Table J-15, several of the components are common to both formulations, and others are unique to each.

Table J-15. Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Neat Conc. In Formulation	Conc. In Treatment[1]	MW (g/mol)	Boiling Pt (^{o}C)	Water Solubility (mg/L @ 25^{o}C)	Vapor Pressure (torr @ 25^{o}C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m^3/mol)	Specific Gravity	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
CFT Legumine																
Rotenone	43,200 mg/L [6.1% by wt of formulation; 4.32% by wt as reported in lab analysis report P-2297, 2298,]	42.1 µg/L	394.4	210-220 / 0.5 mm	0.2 mg/L (Re-registration doc and HSDB)	6.9 x 10^{-10}		1.1 x 10^{-13}	1.27 @ 20^{o}C	4.10	Hydrolysis: 3.2 days @ pH=7, 2 days @ pH=9 Aqueous photolysis: 21 hrs (1 cm), 191 days (2 m, well mixed) Entire pond system (water + sediment): 20 days in cold water (5^{o}C), 1.5 days in warm water (25-27^{o}C) Air photooxidation: 0.05 days Soil: 3 days		TOC: 0.36 mg/kg			LD$_{50}$ Mice (i.p.): 2.8 mg/kg Rats (oral): 132 mg/kg-bw; (i.v.): 6 mg/kg Human: ingestion or inhalation of large doses may lead to: numbness of oral mucosa, respiratory paralysis at lethaldoses, tremor, tachypnea, nausea, vomiting. Chronic exposure may produce fatty changes in liver and kidney. More toxic when inhaled than ingested. Skin irritation from direct contact.
Rotenolone	5,300 mg/L [0.75% by wt of analyzed formulation constituents; 0.53% by wt as reported in lab analyses]	5.2 µg/L	412.42													Oral LD$_{50}$ Mice: rotenolone I, 4.1 mg/kg rotenolone II, 25 mg/kg
1-Methyl-2-pyrrolidinone (Methyl pyrrolidone)	90,000 mg/L [12.71% by wt; 9% by wt as reported in lab analyses]	87.8 µg/L	99.13	202	infinitely soluble in water	0.345	3.4	4.46 x 10^{-8}	< 1.0	-0.54	Air photooxidation: 5 hrs Soil: 4 days in clay, 8.7 days in loam, 11.5 days in sand	1 mg/m^3 = 0.24 ppm	mild amine odor		NOEL = 5 g/L in bacteria, algae (*Scenedesmus)* and protozoa (*Colpoda)*	
Diethylene glycol monoethyl ether (Diethylene glycol ethyl ether)	569,000 mg/L [80.36% by wt; 56.9% as reported in lab analyses]	581.1 µg/L	134.2	202	infinitely soluble in water	0.13	4.62	4.86 x 10^{-8}	0.99 @ 20^{o}C / 4^{o}C	-0.08 (USEPA RAGS E and HSDB)	Air photooxidation: 12 hrs	1 mg/m^3 = 0.188 ppm	Quality: sweet, musty Hedonic tone: unpleasant to pleasant; Abs.: 0.21 ppm 50% recog: 1.10 100% recog: 1.10 O.I. recogn.: 600 O.I. at 20^{o}C = 120	BOD: 0.20 NEN 3235-5.4 COD: 1.85 NEN 3235-3.3	24 hr LC$_{50}$: > 5,000 mg/L (goldfish, static); 96 hr LC$_{50}$: > 10,000 mg/L, (*Menidia beryllina*, static*)*	Oral LD$_{50}$ (single dose): Rat = 8.69-9.74 g/kg Guinea pig: 3.67-4.97 g/kg Cat: 1 ml/kg (lethal) Rat NOEL: 0.49 g/kg (repeat oral dose) Rabbit, cat, guinea pig, mouse inhalation—no injury w/ 12 day exposure to saturated vapor.
1,3,5-Trimethylbenzene (*aka* mesitylene)	4 mg/L [0.00056% by wt]	0.004 µg/L	120.19	164.7	48.2	2.4	1.006 @ 20^{o}C	0.147	0.865	4.00	Aqueous volatilization: est. 3 hrs for model river, 4 days for model lake, and 5 days for model pond (includes sediment adsorption) Air photooxidation: 7 hrs	1 mg/m^3 = 0.203 ppm; 0.4% of emitted hydrocarbons from diesel engines	Avg recog.: 0.027 mg/L Range: 0.00024-0.062 mg/L	BOD: 3% of Theoretical Oxygen Demand (ThOD) COD: 10% of ThOD	96 hr median threshold limit = 13 mg/L (goldfish, flow-through)	

Table J-15. Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Neat Conc. In Formulation	Conc. In Treatment[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
sec-Butylbenzene	3.9 mg/L [0.00055% by wt]	0.004 µg/L	134.21	173	17	1.1 (20°C)	4.62	0.019	0.862		Aqueous volatilization: est. 3.4 hrs for model river, 4.6 days for model lake, and 88 days for model pond (includes sediment adsorption) Air photooxidation: 1.9 days	Relative chemical reactivity [RCR]: 1.31	distinctive aromatic odor			Eye irritation reactivity [EIR] in man @ 1.8
1-Butylbenzene (n-Butylbenzene)	80 mg/L [0.011% by wt]	0.078 µg/L	134.21	183	14	1	4.62	0.0883	0.860	4.03	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake, and 16 days for model pond (includes sediment adsorption) Air photooxidation: 1.8 days	RCR: 1.03		ThOD: 3.22		EIR: 6.4 (man)
4-Isopropyltoluene (p-Isopropyltoluene)	5.1 mg/L [0.00072% by wt]	0.005 µg/L	134	177	16.8	1.75	4.62	0.0183	0.8610 @ 20°C / 4°C	4.16	Aqueous volatilization: est. 1 hr for model river, 5 days for model lake, and 30 days for model pond (includes sediment adsorption) Air photooxidation: 1 day		sweet aromatic odor			
Methylnaphthalene	140 mg/L [0.0198% by wt]	0.136 µg/L	142.19	241	24.6	0.0677	4.91	5.17×10^{-4}	1.025	3.86	Aqueous volatilization: est. 5.5 hrs for model river, 5.3 days for model lake, and 78 days for model pond (includes sediment adsorption) Air photooxidation: 7.4 hrs	1 mg/m³ = 0.17 ppm;	water: 0.023 ppm (range = 0.0025-0.17 ppm) TOC (detection) = 0.0075 mg/kg		24, 48, 72, 96-hr LC_{50} = 39, 9, 9, 9 mg/L in FHM (static); 48-hr LC50 in brown trout yearlings = 8.4 mg/L (static); BCF: 20 to 130 in coho salmon muscle, depending on length of exposure.	

Table J-15. Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Neat Conc. In Formulation	Conc. In Treatment[1]	MW (g/mol)	Boiling Pt (^{o}C)	Water Solubility (mg/L @ 25^{o}C)	Vapor Pressure (torr @ 25^{o}C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m^3/mol)	Specific Gravity	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
Naphthalene	350 mg/L [0.049% by wt]	0.341 μg/L	128.6	217.9	31	0.23	4.42	4.83 x 10^{-4}		3.36	Aqueous volatilization: est. 3 hrs for model river and 5 days for model lake Aqueous photolysis: 71 hrs Aqueous biodegradation: 0.8-43 days Sediment: Degradation rates in sediment are 8-20 times higher than in the above water column. Biodegradation half-lives ranged from 2.4 weeks in sediments chronically exposed to petroleum hydrocarbons to 4.4 weeks in sediment from a pristine environment. Soil biodegradation: 2-18 days Air photooxidation: 18 hrs	1 mg/m^3 = 0.191 ppm	water: 0.021 ppm air: 0.084 ppm			
Noxfish																
Rotenone	50,000 mg/L	48.805 μg/L														
Rotenolone	15,000 mg/L	14.641 μg/L														
Trichloroethene (Trichloroethylene)	73 mg/L	0.071 μg/L	131	87	1,100	75	4.53	0.0103	1.4642 @ 20^{o}C / 4^{o}C	2.71	Aqueous volatilization: est. 3.5 hrs for model river, 5 days for model lake Aqueous hydrolysis: 10.7 months Air photooxidation: 7 hrs	1 mg/m^3 = 0.186 ppm	water: 10 ppm air: 50 ppm, disagreeable above 200 ppm			

Table J-15. Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Neat Conc. In Formulation	Conc. In Treatment[1]	MW (g/mol)	Boiling Pt (°C)	Water Solubility (mg/L @ 25°C)	Vapor Pressure (torr @ 25°C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m³/mol)	Specific Gravity	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
Toluene	1800 mg/L	1.757 µg/L	92.13	110.6	56.2	30	3.1	0.00664	0.8636 @ 20°C / 4°C	2.75	Aqueous volatilization: est. 1 hr for model river and 4 days for model lake Water: 4 days (aerobic), 56 days (anaerobic) Uncontaminated estuarine: 90 days Soil biodegradation: several hrs to 71 days Air photooxidation: 3 days	1 mg/m³ = 0.265 ppm	water: 0.04 ppm air: 2.14 ppm			LD_{50} (rats) 7.53 g/kg
1,3- and/or 1,4-Xylene (M/p xylene)	610 mg/L	0.595 µg/L	106		185	9.5	3.7	0.00766	0.86104 @ 20°C / 4°C	3.20	*1,3-xylene* Aqueous volatilization: est. 3 hrs for model river and 4 days for model lake Air photooxidation: 16 hrs *1,4-xylene* Aqueous volatilization: est. 3 hrs for model river and 4.1 days for model lake Air photooxidation: 27 hrs	1 mg/m³ = 0.23 ppm	mixed isomers: water: 0.53 ppm air: 0.102 ppm			
1,2-Xylene (o xylene)	76 mg/L	0.074 µg/L	106	144	178	7	3.7	0.00519	0.8801 @ 20°C / 4°C	3.13	Aqueous volatilization: est. 3.2 hrs for model river and 4.1 days for model lake Air photooxidation: 1.2 days	1 mg/m³ = 0.23 ppm	mixed isomers: water: 0.53 ppm air: 0.102 ppm			
Isopropylbenzene	52 mg/L	0.050 µg/L	120	153	61.3	4.6	4.1	0.0131	0.862 @ 20°C / 4°C	3.50	Aqueous volatilization: est. 1.2 hrs for model river and 4.4 days for model lake Air photooxidation: 2.5 days		detection: 0.008 ppm recognition: 0.047 ppm			
1-Propylbenzene (n-Propylbenzene)	310 mg/L	0.303 µg/L	120	158	23.4	2.5	4.14	0.00659	0.862 @ 20°C / 4°C	3.60	Aqueous volatilization: est. 1 hr for model river and 4 days for model lake Air photooxidation: 2 days					

Table J-15. Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Neat Conc. In Formulation	Conc. In Treatment[1]	MW (g/mol)	Boiling Pt (^oC)	Water Solubility (mg/L @ 25^oC)	Vapor Pressure (torr @ 25^oC)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m^3/mol)	Specific Gravity	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
1,3,5-Trimethylbenzene	860 mg/L	0.839 µg/L	120.19	164.7	48.2	2.4	1.006 @ 20^oC	0.147	0.865	4.00	Aqueous volatilization: est. 3 hrs for model river, 4 days for model lake, and 5 days for model pond (includes sediment adsorption) Air photooxidation: 7 hrs	1 mg/m^3 = 0.203 ppm; 0.4% of emitted hydrocarbons from diesel engines	Avg recog.: 0.027 mg/L Range: 0.00024-0.062 mg/L;	BOD: 3% of Theoretical Oxygen Demand (ThOD) COD: 10% of ThOD	96 hr median threshold limit = 13 mg/L (goldfish, flow-through)	
1,2,4-Trimethylbenzene	10,000 mg/L	9.761 µg/L	120	169	57	2.1	4.15	0.00616	0.8761 @ 20^oC / 4^oC	3.78	Aqueous volatilization: est. 3 hrs for model river and 4 days for model lake Air photooxidation: 12 hours	1 mg/m^3 = 0.203 ppm				
1-Butylbenzene (n-Butylbenzene)	9,000 mg/L	8.785 µg/L	134	183	14	1	4.62	0.0883	0.860	4.03	Aqueous volatilization: est. 3.5 hrs for model river, 4.6 days for model lake, and 16 days for model pond (includes sediment adsorption) Air photooxidation: 1.8 days					
4-Isopropyltoluene (p-Isopropyltoluene)	1,000 mg/L	0.976 µg/L	134	177	16.8	1.75	4.62	0.0183	0.8610 @ 20^oC / 4^oC	4.16	Aqueous volatilization: est. 1 hr for model river, 5 days for model lake, and 30 days for model pond (includes sediment adsorption) Air photooxidation: 1 day		sweet aromatic odor			

Table J-15. Physical and Chemical Properties of Rotenone Formulation Constituents

Ingredient	Neat Conc. In Formulation	Conc. In Treatment[1]	MW (g/mol)	Boiling Pt (^{o}C)	Water Solubility (mg/L @ 25^{o}C)	Vapor Pressure (torr @ 25^{o}C)	Vapor Density (Vd = PM/RT)[2]	Henry's Law Constant (atm-m^3/mol)	Specific Gravity	Log Octanol/Water Partition Coefficient	Half-Lives[3]	Air Pollution Factors[4]	Odor Thresholds and Characteristics	Water Pollution Factors	Aquatic Toxicity Metrics	Toxicity to Other Receptors
Naphthalene	70,000 mg/L (EPA method 8260) 28,000 mg/L (EPA method 8270)	68.326 µg/L (w/ EPA 8260)	128	217.9	31	0.23	4.42	4.83 x 10^{-4}	1.162	3.36	Aqueous volatilization: est. 3 hrs for model river and 5 days for model lake Aqueous photolysis: 71 hrs Aqueous biodegradation: 0.8-43 days Sediment: Degradation rates in sediment are 8-20 times higher than in the above water column. Biodegradation half-lives ranged from 2.4 weeks in sediments chronically exposed to petroleum hydrocarbons to 4.4 weeks in sediment from a pristine environment. Soil biodegradation: 2-18 days Air photooxidation: 18 hrs	1 mg/m^3 = 0.191 ppm	water: 0.021 ppm air: 0.084 ppm			
Potassium Permanganate Rotenone Neutralization Compound																
Potassium permanganate (neutralizing compound for lake outlet treatment)	4 mg/L-water (maximum neutralization concentration)		158		64,000 (20^{o}C)	na	na	na	na				odorless		96-hr LC_{50}: 3.6 mg/L (goldfish) 0.75 mg/L (channel catfish) 96-hr LD_{50}: 2.7-3.6 mg/L (bluegill)	Oral LD_{50} (single dose): Guinea pig: 810 mg/kg Mouse: 750 mg/kg Rat: 750 mg/kg

J.3.6.1.1 CFT Legumine®

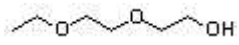
The two primary inactive carrier components in CFT Legumine® are 1-methyl-2-pyrrolidinone and diethylene glycol monoethyl ether, which comprise approximately 93% of the formulation by weight of the constituents that were identified in the analysis conducted by the DFG (Table J-15). Both of these chemicals are infinitely soluble in water and have an estimated organic carbon partition coefficient (i.e., the "K_{oc}") of 12, indicating that they will remain in the water column and will not tend to adsorb to sediment particulates (NLM, 2006). Based on their low Henry's Law constants, these chemicals will not readily volatilize from surface water, and neither chemical is expected to undergo hydrolysis or direct photolysis (NLM, 2006).

Aerobic biodegradation is expected to be the most important mechanism for the removal of 1-methyl-2-pyrrolidinone and diethylene glycol monoethyl ether from aquatic systems (NLM 2006). The small amount of these chemicals that may volatilize into ambient air will be readily degraded by reaction with photochemically-produced hydroxyl radicals, with an atmospheric half-life of up to 12 hours (NLM 2006). The remaining carrier chemicals include the polycyclic aromatic hydrocarbons (PAH) naphthalene and methylnaphthalene and a few alkylated benzenes. While these chemicals are more volatile than the primary carriers, they comprise less than one percent of the formulation and are not expected to significantly impact the overall fate and transport of CFT Legumine.

The structures and oral toxicities of the three most concentrated constituents in CFT Legumine are summarized below.

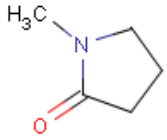
Diethylene glycol monoethyl ether

- Approximate concentration in formula: 569,000mg/L
- Toxicology: RAT ORAL LD_{50}: 4700-9740mg/kg.
- Chemical formula: $C_6H_{14}O_3$
- Chemical structure: $C_2H_5OCH_2CH_2OCH_2CH_2OH$



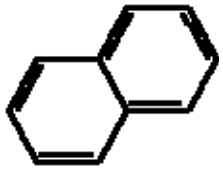
1-Methyl-2-Pyrrolidinone

- Approximate concentration in formula: 90,000mg/L
- Toxicology: RAT ORAL LD_{50}: 3914 mg/kg
- Chemical formula: C_5H_9NO



Naphthalene

- Approximate concentration in formula: 350mg/L
- Toxicology: MOUSE ORAL LD_{50}: 533mg/kg
- Chemical formula: $C_{10}H_8$
- Chemical structure:

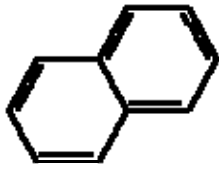


J.3.6.1.2 Noxfish®

In contrast to CFT Legumine, the inert and carrier chemicals for Noxfish® consist of the PAH naphthalene, numerous alkylated benzenes, and trichloroethene. These chemicals are moderately soluble in water, with aqueous solubilities ranging from 14 to 1,100 mg/L (NLM, 2006). K_{oc} values range from 94 to 3,200 L/kg, suggesting that these chemicals may also tend to adsorb to sediment particulates, thus increasing their half-lives in natural waterbodies (NLM, 2006). The half-lives for these chemicals in surface water bodies range from several hours to several months, depending on the characteristics of the waterbody (i.e., temperature, flow velocity, agitation, etc.), as well as the amount of sunlight on the water surface. With Henry's Law constants ranging from 0.00048 to 0.15 atm-m^3/mol, the primary removal mechanism from surface water for these carrier chemicals is volatilization, with direct photooxidation, hydrolysis and biodegradation contributing to a much smaller degree. Once in the ambient air, chemical vapors are readily degraded by reaction with photochemically-produced hydroxyl radicals. The chemical-specific half-lives for this reaction in air range from a few hours to a few days (NLM, 2006). Of particular note is naphthalene, which comprises slightly less than 50% of the NoxFish formulation by weight of the constituents identified in the analysis provided in Table J-15. This PAH, which gives moth balls their distinctive odor, has an odor threshold in air of 0.084 ppm, or 0.44 mg/m^3.

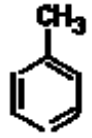
Naphthalene

- Approximate concentration in Noxfish® formula: 70,000 mg/L
- Toxicology: MOUSE ORAL LD_{50}: 533 mg/L
- Chemical formula: $C_{10}H_8$
- Chemical structure:



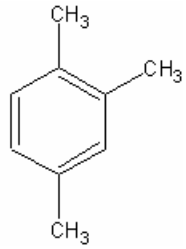
Toluene

- Approximate concentration in Noxfish® formula: 1,800 mg/L
- Toxicology: MOUSE ORAL LD_{50}: 636 mg/kg
- Chemical formula: C_7H_8
- Chemical structure:



1,2,4 Trimethylbenzene

- Approximate concentration in Noxfish® formula: 10,000 mg/L
- Toxicology: MOUSE ORAL LD_{50}: 5,000 mg/kg
- Chemical formula: C_9H_{12}
- Chemical structure:



J.3.6.1.3 Powdered Rotenone

The inert ingredients in the powdered ('cube root') rotenone product are plant fiber from the root of the plants ground up to produce the product (Finlayson et al. 2000 as cited in WDFW 2002). The plant fiber constitutes approximately 81.5% of the powder form of rotenone while 11.1% is associated with plant resins and 7.4% is active rotenone (WDFW 2002). Because of the low application rates required for rotenone used in fisheries management, the entire plant root is ground up and packaged rather than extracting and/or concentrating the active chemical rotenone from the ground up roots.

J.3.6.2 Fate, Transport and Toxicity of Proposed Rotenone Formulation Constituents and Potassium Permanganate Neutralization Solution

J.3.6.2.1 Review of Rotenone Dispersant Fate and Toxicity from Field Studies Conducted Outside Project Area

California researchers have monitored surface and ground water in nine projects in California lakes and streams treated with liquid rotenone formulations and powdered rotenone formulations since 1987 (CDFG 2001). They determined that all the measured concentrations of dispersant ingredients were well below the minimum concentrations allowed for drinking water standards developed by USEPA. For example, these researchers found that concentrations of TCE never exceeded the USEPA drinking water standard (Maximum Contaminant Level) of 5 μg/L and similarly the concentrations of xylene never exceeded the drinking water standard (Health Advisory) of 620 μg/L (WDFW 2002). Drinking water standards for naphthalene and methylnaphthalenes have not been established, however, the researchers found that these volatile organic compounds (VOCs) and semivolatile organic compounds (SVOCs) disappeared before rotenone dissipated, typically within 1 to 3 weeks.

The physico-chemical properties of the VOCs and SVOCs in the rotenone formulations similarly do not lend themselves to appreciably accumulate or persist in sediment. In the Finlayson et al. (2001) report, rotenone, rotenolone and some semi-VOC (napthalene and methylnapthalene) were detected above the analytical detection limits of 30 micrograms/kg-dry wt for rotenone and rotenolone, and 6 ug/kg for the volatile and semivolatile organic compounds. In standing water sediments from these nine study sites rotenone and rotenolone were detected a maximum of 60 days, with maximum concentrations of 522 and 890 ug/kg-dry wt, respectively. No VOCs (e.g., xylene, TCE) were ever detected, in either flowing or static water sediments. The only semi-VOCs detected in lake sediments were naphthalene, 1-methylnaphthalene, and 2-methylnapthalene. Detectable concentrations of these semi-VOCs were measured up to 180 days after treatment in standing water sediments, with maximum concentrations of 91 and 231 ug/kg for napthalene and methylnapthalene, respectively.

Based on information collected related to rotenone formulation applied previously to Lake Davis, California, several VOCs and SVOCs were associated with the formulated end-product used (USEPA, 2006). These chemicals included naphthalene, methyl naphthalene, toluene and xylene. Additionally, TCE, a contaminant from the extraction of rotenone from plant tissues has also been reported. In addition to these compounds, formulated end-products may also contain varying amounts of cube root resin (rotenoloids such as rotenololone) and the extent of their toxicity is uncertain. However, toxicity testing with formulated end-products suggest that in general, co-formulants do not substantially affect the toxicity of rotenone based on reported distributions of acute 96-hr LC_{50} values amongst diferent species (USEPA 2005). Based on these results it is assumed that the distribution of species sensitivities observed from laboratory tests represent the distribution of sensitivities that are likely to be encountered in the environment.

The Minnesota Department of Health conducted a risk assessment of the inert ingredients in Nusyn-Noxfish® (used in the 1997 Lake Davis treatment, but not a formulation considered currently) for the Minnesota Department of Natural Resources. Their assessment reported

August 7, 1991, stated that "There is negligible risk to human health from the contaminants found in rotenone whether the exposure is from drinking, swimming or eating fish from treated waters (*as cited in* WDFW 2002). In addition, they determined that treatment with rotenone will introduce contaminants into the lake, but at concentrations considerably lower than the levels that would harm human health" (WDFW 2002).

As part of the re-registration process USEPA (2006) conducted a review of the available toxicity data on all of the formulated products of rotenone for all of the surrogate species typically evaluated, however, only limited toxicity data were available on the inert ingredients. In reviewing the toxicity data collected on both technical grade rotenone (>95% active ingredient) and formulated end-product, USEPA (2006) determined that the technical grade active ingredient is generally more toxic than formulated end-product [corrected for active ingredient] by at least a factor of two. These data suggest that for the formulated products tested and the toxicity endpoints measured, the dispersant ingredients do not contribute substantially to the toxicity of the active ingredient and are effectively inert.

In addition, USEPA (2006) indicated that these data also suggest that the similarly structured rotenolones of plant resins (cube root resins) contained in varying amounts in formulated end-products also do not contribute substantially to the toxicity of rotenone. Rotenolone persists longer than rotenone, especially in cold, alpine lakes; rotenolone has been detected for as long as 6 weeks in cool water temperatures (<10°C) at high elevations (>8,000 feet). In part, this situation occurs because rotenone may be more susceptible to photolysis than rotenolone (Finlyason et al, 2000). However, studies have indicated that rotenolone is approximately one-tenth as lethal as rotenone (DFG 1991a as cited in Finlayson et al, 2000). In those rare cases of rotenolone persistence, fish stocking would be delayed until both rotenone and rotenolone residues have declined to nondetectable (<2 ppb) levels to err on the side of safety (Finlayson et al, 2000). Table J-16 summarizes available toxicity information on inert ingredients identified in the rotenone formulations proposed for use.

J.3.6.2.2 Review of Environmental Fate Findings from Past Rotenone Treatment of Lake Davis

The following discussion briefly examines monitoring results in environmental media from past rotenone treatments. Such data provide additional perspective on the environmental fate of some of the components in the rotenone formulations under current consideration.

Surface Water

Sample results from the last treatment at Lake Davis indicate that both Rotenone and Rotenolone surface water residues declined to below the detection limits (2 μg/L) 48 days following application (Siepmann & Finlayson 1999). Comparatively, the results demonstrated that most of the dispersant compounds dissipated before rotenone. Within a week of the treatment, VOC residues were completely absent from the samples while the semi-VOCs persisted for no longer than two weeks. The only compound that remained in Lake Davis surface water long after the dissipation of rotenone, a total of thirty-nine weeks post-treatment, was PBO. Again, PBO is not found in any of the formulations under consideration for use.

Table J-16. Aquatic and Terrestrial Toxicity Information on Inert Ingredients Identified in the Rotenone Formulations Proposed for Use in Lake Davis

Ingredient	Toxicity to Aquatic Receptors	Toxicity to Terrestrial Receptors			
		Acute ORAL LD_{50}	IHL LC_{50} /IPR/IVN LD_{50}	Acute Dermal LD_{50}	Other
Rotenone	See rotenone information				
Rotenolone	Not Available	Not Available	Not Available	Not Available	Not Available
Methyl pyrrolidone (*aka* n-methylpyrroli)		RAT: 3914 mg/kg MUS: 7725 mg/kg	IPR-RAT LD_{50}: 2472 mg/kg IVN-RAT LD_{50}: 2266 mg/kg	RBT: 8000 mg/kg	Typical LTEL: 25 ppm. AIHA Workplace environmental exposure level: 10ppm (8h).
Diethylene glycol ethyl ether	24h LC50: 5,000 mg/L (Goldfish, static). 96h LC_{50}: >10,000 mg/L (*Menidia beryllina*, static)	RAT: 8690-9740 mg/kg GPIG: 3670-4970 mg/kg			CAT: 1 ml/kg (lethal) RAT NOEL: 490 mg/kg (repeat oral dose) RBT,CAT,GPIG,MUS inhalation: no injury with 12d exposure to saturated vapor.
1,3,5 trimethylbenzene (aka mesitylene)			IHL-RAT: 24 mg/m^3 (4h)		Typical STEL: 35 ppm.
Sec-butylbenzene			IHL-RAT: >1900 mg/kg	RBT: >13000 mg/kg	Eye irritation reactivity [EIR] in MAN @ 1.8
n-butylbenzene	Unknown	Unknown	Unknown	Unknown	EIR in MAN: 6.4
p-isopropyltoluene (*aka* p-cymene)		RAT: 3669-4750 mg/kg	IHL-MUS: 19500 mg/m^3		RBT (Moderate skin irritation): 500 mg (24h).
Methyl napthalene (*aka* 1-Methylnapthalene)	24,48,72,96h LC_{50}: 39,9,9,9 mg/L in FHM (static). 48h LC_{50}: 8.4 mg/L in B.trout yearlings (static). BCF:20-130 in Coho salmon muscle, depending on exposure time.	RAT: 1840 mg/kg			RBT-SKIN-LDLO (lowest recorded lethal dose): 7500mg/kg.

Table J-16. Aquatic and Terrestrial Toxicity Information on Inert Ingredients Identified in the Rotenone Formulations Proposed for Use in Lake Davis

Ingredient	Toxicity to Aquatic Receptors	Toxicity to Terrestrial Receptors			
		Acute ORAL LD_{50}	IHL LC_{50} /IPR/IVN LD_{50}	Acute Dermal LD_{50}	Other
Napthalene	96h LC_{50}: 305.2 ppm (Trout)	MUS: 533 mg/kg RBT: 3000 mg.kg	IVN-MUS: 100 mg/kg		LDLO (lowest published lethal dose) for Child: 100mg/kg (ORAL) LDLO for human: 29 mg/kg (unknown entry). Threshold Limit Value (TLV): 10 ppm. RBT (Mild skin irritation): 100 mg. RBT (Mild eye irritation): 495 mg.
n-methyl-2-pyrrolidone	See Tox data for Methyl Pyrrolidone				
Di ethyl ether		RAT: 1215 mg/kg MAN-LDLO: 260 mg/kg	IHL-MUS: 31000 ppm (0.5h)		Human eye irritation: 100 ppm. RBT (Mild Skin irritation): 360 mg GPIG (Severe skin irritation): 30 mg/24h.
Ethylene glycol		RAT: 4700 mg/kg HUMAN-LDLO: 786 mg/kg	IPR-MUS: 5614 mg/kg		
Trichloroethylene		RAT: 7193 mg/kg HUMAN-LDLO: 7000 mg/kg	IPR-DOG: 1900 mg/kg IVN-MUS: 34 mg/kg IHL-HUMAN-TCLO: 6900 mg/m^3 (10 mins) (Lowest Published Toxic Concentration). IHL-MAN-LCLO: 2900 ppm		Typical STEL: 150 ppm Typical LTEL: 100 ppm

Table J-16. Aquatic and Terrestrial Toxicity Information on Inert Ingredients Identified in the Rotenone Formulations Proposed for Use in Lake Davis

Ingredient	Toxicity to Aquatic Receptors	Toxicity to Terrestrial Receptors			
		Acute ORAL LD_{50}	IHL LC_{50} /IPR/IVN LD_{50}	Acute Dermal LD_{50}	Other
Toluene		RAT: 636 mg/kg RAT: 2600-7500 mg/kg HUMAN-LDLO: 50 mg/kg	IPR-RAT: 1332 mg/kg IPR-MUS: 59 mg/kg IHL-RAT: 8000 ppm (4h) IHL-Unspecified Mammal species: 30g/m^3		RBT (Mild Skin irritation): 435 mg. Human eye irritation: 300 ppm.
Ethylbenzene	LC_{50} (96h): Trout: 4.2mg/L FHM: 12.1mg/L Guppy: 9.9mg/L Bay Shrimp: 0.490mg/L Crab: 13mg/L	RAT: 3500 mg/kg	IHL-GPIG-LCLO: 10000 ppm.	RBT: 17800 mg/kg	RBT (Mild Skin irritation): 15 mg (24h).
M xylene		RAT: 5000 mg/kg			Typical PEL (prolonged exposure limit): 100 ppm.
P xylene		RAT: 5000 mg/kg	IPR-RAT-LDLO: 2000 mg/kg		Typical PEL (prolonged exposure limit): 100 ppm
O xylene		RAT: 4000 mg/kg	IPR-MUS: 1.5 ml/kg		Typical STEL: 150 ppm
Isopropyl benzene (*aka* cumene/cumol)		RAT: 1400 mg/kg	IHL-RAT: 8000 ppm (4h)	RBT: 12300 mg/kg	Typical TLV/TWA: 50 ppm
n-propylbenzene (*aka* propylbenzene)		RAT: 6040 mg/kg			
1,2,4-trimethylbenzene		RAT: 5000 mg/kg	IHL-MUS: 8147 ppm IPN-RAT-LDLO: 2000 mg/kg IPN-GPIG-LDLO:1566 mg/kg		

Sediment

Past monitoring of sediment quality following the 1997 Lake Davis treatment (Siepmann and Finlayson 1999) is generally reflective of the broader results discussed above. Specifically:

- The measured levels of rotenone and rotenolone in lake bottom sediments had dropped below detection limits 55 days after treatment;
- No VOCs were detected in sediment samples; and
- Semi-VOCs (naphthalene, 1-methylnaphthalene, and 2-methylnaphtalene) were detected in sediment samples, but measured levels of these compounds dropped below detection limits 55 days after treatment.

Groundwater

Imperical evidence does not support that groundwater contamination could be expected from the proposed use of either rotenone formulation or from the powdered form considered for use under Alternative A. Post treatment groundwater monitoring by the DFG in 26 wells from nine areas where Nusyn-Noxfish® had been applied, including five wells in the 1997 Lake Davis treatment area, failed to identify groundwater contamination with VOC and SVOCs in wells monitored up to 456 days following treatment—with the exception of a single xylene detection in a Corps of Engineers well 59 days after treatment of the Kaweah Reservoir. Notably, all of the five wells monitored by DFG were located immediately adjacent to the reservoir, and four were at the southern end, downgradient of the reservoir's outlet. Given groundwater flows "downhill," it is reasonable to assume the groundwater quality in these wells would have been affected if significant groundwater mobility of the rotenone formulation constituents occurred.

The DFG data are consistent with the ongoing 10-year monitoring program being conducted by Plumas County Environmental Health, where 81 wells in the Lake Davis project area have been monitored for potential contamination following the 1997 treatment. In the PCEH monitoring to date, there was a verified detection of toluene and an inconsistent and unverified detection of trichloroethylene. Thse detections were below MCLs and did not show any spatial or temporal pattern that might suggest the 1997 application as the source (see Section 4, Table 4.2-1).

It was concluded that the detection of these solvents, common to pump apparatus, fuels, and dry cleaning solvents, was not likely attributed to the 1997 lake treatments, given the well locations and the transient nature of detections (usually one detection event only per well). Perhaps more importantly, as summarized in Section 4 of the EIR/EIS, current understanding suggests that groundwater is a highly unlikely pathway for exposure to the surrounding community because: (1) City of Portola wells tap into a deeper aquifer that is distinct from Lake Davis, (2) groundwater generally discharges into Lake Davis (not the other way around), (3) private wells downgradient of Lake Davis principally recharge from the east and west of the Big Grizzly Creek watershed and have not been shown to have connectivity to lake levels in monitoring conducted by the Department of Water Resources, and (4) the

nearest private wells are over 1,000 feet from Lake Davis, which would require an extensive migration for contaminants to be detectable yet whose volatility and non-persistence indicate rapid degradation is likely.

Air

Following the previous rotenone treatment of Lake Davis in 1997, between October 24 and October 29, seventy one residents near the project area reported symptoms potentially associated with the use of the Nusyn-Noxfish® rotenone formulation (CEPA 1998). Sixty-seven of these individuals were interviewed by staff from the Pesticide Illnes Surveillance Program (PISP), and 60 reported smelling strong hydrocarbon odors. Adverse health symptoms reported included eye irritation, upper respiratory irritation, and other non-specific systemic symptoms (e.g., nausea, headache, diarrhea, wheezing). Reports were particularly consistent for symptoms of ocular and mucous membrane irritation from individuals that were present at the lake or dam over both days of application (there was no public closure of the project area during treatment).

Based on the initial complaints to the Department of Pesticide Regulation (DPR) on October 16, a sampling program was initiated by the Air Resources Board emergency response staff on October 18. In this sampling program the air board set up six stations near and around the lake to monitor for rotenone and aromatic hydrocarbons that may be have been emitted into the air from the treatment. Air samples were collected by integrated charcoal tube sampling. Samples were collected at the dam face, at the spillway below the dam face, at a nearby campground, and three stations downstream. Heavy aromatic hydrocarbons (primarily napthalene, and methyl napthalene), light hydrocarbons (e.g., xylene, benzenes, toluene), TCE, and rotenone were measurable at the highest concentrations at the spillway below the dam. Principal findings included:

- TCE was measured at 0.5 ppm (12-hr average) at the spillway on October 18 but all other lighter hydrocarbons (e.g., xylene, benzene) were below detection limits of 3 ppb (by volume).
- Total heavy hydrocarbons measured at the spillway peaked at 1740 ug/m^3 on October 18th, in the spillway below the dam. It is estimated from analysis of the composition of a similar air sample that approximately 15.5% of the hydrocarbon content was contributed by napthalene (269 ug/m^3). Total hydrocarbon concentrations were reduced to 10 ppb by November 1.
- Rotenone was measurable above detection limits at the spillway site below the dam until October 30th. Unlike the hydrocarbon measurements, rotenone concentrations exhibited less variability from day to day, with the peak concentration (0.53 ppb) actually measured 4 days after the dispersion of the rotenone formulation into the lake had been completed. The maximum value of rotenone empirically detected was nearly an order of magnitude lower than the no effect inhalation value specified by the Office of Environmental health and Hazard Assessment (OEHHA) and the Department of Pesticide Regulation for 24-hr average exposure (430 ppb). Notably, Screen3 modeling of projected air concentrations of rotenone, assuming worst case conditions, estimated a concentration of 1 ppm,

- Air concentrations of all measured constituents, including the 'bridge' site 100 yards downstream of the spillway, was considerably lower or non-detectable at all other sites than at the spillway, including the 'bridge' site 100 yards downstream of the spillway, as would be expected given the disruption of the water:air interface created at the spillway.
- Based on Screen3 dispersion modeling of potential rotenone and total heavy hydrocarbons, it was estimated that air concentrations would not exceed permissable exposure levels (PELs) applicable to occupational criteria.

J.3.6.2.3 Potassium Permanganate Neutralizing Solution

Potassium permanganate ($KMnO_4$) is a strong oxidizing agent used in many industries and laboratories. It is also used as a disinfectant, especially in the treatment process of potable water. In fisheries and aquaculture, $KMnO_4$ is utilized as a treatment for some fish parasites. Under the Proposed Project, it is and can be used as a neutralizing compound following the addition of rotenone to a body of water (EPA, 2006; Ling, 2003). Following rotonone application, after a crucial time interval based on the management goal of the fishery, $KMnO_4$ is added to the water at ratios of between 2 and 4 parts $KMnO_4$ to each part of rotenone (EPA, 2006). Under the Proposed Project, this concentration may approximate 4 ppm, depending on the organic load in the receiving water at the time of treatment.

Manganese is the principal element in the permanganate solution with potential toxicity. However, manganese is also an essential nutrient for plants and animals, and specific deficiency signs have been identified with a wide range symptoms including nervous system disorders, bone fragility, and growth suppression (Browning1961). Manganese comprises about 0.1% of the earth's crust and is ubiquitous in the environment (rock, soil, water). Potassium permanganate is produced by thermal oxidation of manganese dioxide (MnO_2) followed by electrolytic oxidation. The environmental chemistry and fate of manganese is controlled largely by pH. At pH values above 5.5 (approximately), colloidal manganese hydroxides generally form in water. Such colloidal forms are not generally bioavailable. As a strong oxidizing agent, permanganate is reduced when it oxidizes other substances (such as rotenone). Thus, in the process of oxidizing rotenone, the $KMnO_4$ is itself reduced, liberating bioavailable oxygen in the process. Through this mechanism, the respiratory toxicity caused by rotenone is effectively countered. In the process, potassium ions are liberated (also essential electrolyte), and manganese dioxide is formed. Manganese dioxide is insoluble, hence not bioavailable, and chemically similar to the MnO_2 found in the earth's crust (Vella 2006).

In the presence of rotenone (and other organic reducing agents for that matter), permanganate will be reduced, will not persist in the environment, and poses essentially no human health risk to groundwater quality. Indeed, it is used second only to chlorine as a pre-treatment method for the removal of organic contaminants such as napthalene and tetrachloroethene (TCE) in potable groundwater wells according to a recent survey by the American Water Works Association (*as cited in* Vella 2006). In groundwater, its use helps to control iron, manganese, sulfides and color, and it can also be used to reduce high concentrations of radionuclides and arsenic (again, by forming insoluble colloids). Potassium permanganate is also used in surface water treatment plants, primarily for taste and odor problems.

Like rotenone, the aquatic toxicity of $KMnO_4$ differs among species. Because of the volume of $KMnO_4$ that may be required for neutralization (i.e., depending on which option is selected), and its moderate to high toxicity to fishes, this neutralizing compound may itself present a hazard to aquatic vertebrates during application. It has been reported to elicit toxicity at concentations of 1 to 2 ppm (EPA, 2006). However, this toxicity range also lies within its therapeautic range for fish disease therapy. Indeed therapeautic doses range from 2 to 25 ppm, depending on the time prescribed for treatment (i.e., prolonged bath versus dip treatments). A concentration of 4 ppm is generally recommended for "permanent bath" treatments of external parasites (Cross and Needham 1988). In a permanent bath, no flushing is anticipated and degradation is through natural oxidative processes—generally occuring within 1 to 4 days. Marking & Bills (1975) demonstrated that its toxicity was inversely proportional to water temperature for both rainbow trout and channel catfish. It is reported to be more toxic in hard water, due to potential precipitation of manganese dioxide on fish gills. Although not as well studied, $KMnO_4$ is also considered to be toxic to aquatic invertebrates and zooplankton although, as with vertebrates, there is likely to be a wide tolerance range between various freshwater invertebrates.

J.3.6.2.4 Regulatory Screening Values for Carrier and Dispersant Ingredients in Rotenone Formulations, and Potassium Permanganate Neutralizing Agent

Regulatory screening values reflect promulgated standards (i.e., numeric criteria) based on a thorough review of available literature, and consensus among the USEPA in the evaluation of the effects literature associated with the exposure pathway. These standards undergo extensive public review through NEPA prior to being adopted as regulatory standards. Such criteria, with their foundations in the toxicological literature, can serve as toxicity reference values (TRVs) by which to gauge risks from environmental exposure to chemicals in air, water or sediment However, for many chemicals, no federal or state regulatory criteria exist, and TRVs must be derived for which no numeric criteria have been promulgated. This section discusses the range of regulatory criteria that have been established for the rotenone formulation constituents identified in Table J-15, and for potassium permanganate, the proposed neutralizing agent.

For the chemical constituents in the rotenone formulations proposed for use, Federal and state regulatory criteria have been identified by the EPA and under the California Toxics Rule (however, these standards are only applicable to human health assessment endpoints, and no surface water, freshwater sediment, soils, or air criteria for the protection of aquatic life (or terrestrial ecological receptors) are promulgated in the CTR or other federal or state regulation.

Table J-17, Table J-18, Table J-19, and Table J-20 summarize these human health regulatory standards for groundwater, surface water, air, and soils, respectively. These regulatory criteria and guidance levels are useful for screening human health risks, by comparing them to the estimated environmental concentration in the different media to which receptors could be exposed to chemicals of concern (e.g., water, air, soil) from the Proposed Project and alternatives. However, it should be noted that the criteria identified in Table J-17, Table J-18,

Table J-19, Table J-20, and Table J-21 are designed and intended to apply to exposure periods that far exceed the duration of exposure to formulation constituents that would be possible for human and ecological receptors from the Proposed Project and alternatives. All but one of the criteria listed in Table J-17, Table J-18, Table J-19, and Table J-20 are based on chronic (long-term) continuous exposure, whereas both human and ecological exposures to the rotenone formulation constituents under the Proposed Project and alternatives will not exceed subchronic duration, and exposure to most constituents will be short term.

Tables J-17 and J-18 present taste and odor threshold values for some compounds in water. These concentrations are taken from regulatory guidance and scientific literature. The taste threshold concentration is the lowest concentration in water that results in the ability of a person to identify a taste and/or odor associated with that compound in water. Table J-19 presents the lowest concentration of chemicals in air (threshold) where an odor is identifiable. In some cases, the health protective concentration in the medium (air or water) is higher than the odor threshold. In such cases, odor provides a signal that can be used to be health protective. However, for other chemicals, the health protective concentration is lower than the odor threshold. In those cases, odor is not an adequate indicator or warning of potential health risk.

Only the California surface water value for the 30-day average concentrations protective of human health for ingestion of water and consumption of aquatic organisms actually addresses a relevant exposure period for the project. This value is also conservative, since it assumes that the exposed human population uses the water for drinking and for the source of fish for their diet for the 30 days. Neither of these exposure pathways is actually likely to be complete for the project, but the criteria provide perspective on the projected concentrations of rotenone and other formulation compounds.

Table J-17. Groundwater Regulatory Values and Odor Thresholds for Rotenone and Other Components of CFT Legumine and NoxFish

Component	Formulation		USEPA Region IX Groundwater PRG (mg/L)	Maximum Contaminant Levels [CW] (mg/L)		Secondary Maximum Contaminant Levels [CW] (mg/L)		California Action Level [CW] (mg/L)	California PHG [CW] (mg/L)	Taste and/or Odor Threshold in Water (mg/L)
	CFT Legumine	NoxFish		Cal/EPA	USEPA	Cal/EPA	USEPA			
Rotenone	√	√	0.15	-	-	-	-	-	-	-
Volatile Formulation Components										
Butylbenzene, 1-	√	√	0.24	-	-	-	-	0.26	-	-
Butylbenzene, sec-	√		0.24	-	-	-	-	0.26	-	-
Isopropylbenzene		√	0.66	-	-	-	-	0.77	-	0.0008 [CW]
Isopropyltoluene, 4-	√	√	0.72	-	-	-	-	-	-	-
Methylnaphthalene, 2-	√		0.0062	-	-	-	-	-	-	0.023 [H]
Naphthalene	√	√	0.000093	-	-	-	-	0.17	-	0.021 [CW, H]
Propylbenzene, 1-		√	0.24	-	-	-	-	0.26	-	-
Toluene		√	0.72	0.15	1	-	0.04	-	0.15	0.042 [CW]
Trichloroethene		√	0.0014	0.005	0.005	-	-	-	0.0008	0.31 [CW]
										10 [H]
Trimethylbenzene, 1,2,4-		√	0.012	-	-	-	-	0.33	-	-
Trimethylbenzene, 1,3,5-	√	√	0.012	-	-	-	-	0.33	-	0.015 [CW]
										0.027 [H]
Xylene, 1,2- [1]		√	0.21	1.75	10	-	0.02	-	1.8	0.017 [CW]
										0.53 [H]
Xylene, 1,3- and/or 1,4- [1]		√	0.21	1.75	10	-	0.02	-	1.8	0.017 [CW]
										0.53 [H]
Semivolatile Formulation Components										
Diethylene glycol monoethyl ether	√		2.2	-	-	-	-	-	-	0.021
Methyl-2-pyrrolidinone, 1-	√		-	-	-	-	-	-	-	-
Rotenolone	√	√	-	-	-	-	-	-	-	-

- = not available.
PRG = preliminary remediation goal for chronic exposure (USEPA, 2004); based on target HI = 1 or target risk = 1x10-6.
a Values apply to mixed xylenes.
CW California Water Quality Goals, 8/2003.
H Hazardous Substances Data Bank, 4/2006.

Table J-18. Surface Water Regulatory Values and Odor Thresholds for Rotenone and Other Components of CFT Legumine and NoxFish

Component	Formulation		California Toxics Rule for Inland Surface Waters; Drinking Water + Consumption of Aquatic Organisms; 30-day Avg [CW] (mg/L)	USEPA AWQC for Drinking Water Consumption of Aquatic Organisms[CW] (mg/L)	Taste and/or Odor Threshold in Water (mg/L)
	CFT Legumine	NoxFish			
Rotenone	√	√	-	-	-
Volatile Formulation Components					
Butylbenzene, 1-	√	√	-	-	-
Butylbenzene, sec-	√		-	-	-
Isopropylbenzene		√	-	-	0.0008 [CW]
Isopropyltoluene, 4-	√	√	-	-	-
Methylnaphthalene, 2-	√		-	-	0.023 [H]
Naphthalene	√	√	-	-	0.021 [CW,H]
Propylbenzene, 1-		√	-	-	-
Toluene		√	6.8	1.3	0.042 [CW]
Trichloroethene		√	0.0027	0.0025	0.31 [CW]
					10 [H]
Trimethylbenzene, 1,2,4-		√	-	-	-
Trimethylbenzene, 1,3,5-	√	√	-	-	0.015 [CW]
					0.027 [H]
Xylene, 1,2- [a]		√	-	-	0.017 [CW]
					0.53 [H]
Xylene, 1,3- and/or 1,4- [a]		√	-	-	0.017 [CW]
					0.53 [H]
Semivolatile Formulation Components					
Diethylene glycol monoethyl ether	√		-	-	0.021
Methyl-2-pyrrolidinone, 1-	√		-	-	-
Rotenolone	√	√	-	-	-

- = not available.

[a] Values apply to mixed xylenes.

[CW] California Water Quality Goals, 8/2003.

[H] Hazardous Substances Data Bank, 4/2006.

Table J-19. Ambient Air Regulatory Values and Odor Thresholds for Rotenone and Other Components of CFT Legumine and NoxFish

Component	Formulation		USEPA Region IX Ambient Air PRG (mg/m^3)	California Acute REL (mg/m^3)	Odor Threshold in Air (ppm)
	CFT Legumine	NoxFish			
Rotenone	√	√	0.015	-	-
Volatile Formulation Components					
Butylbenzene, 1-	√	√	0.15	-	-
Butylbenzene, sec-	√		0.15	-	-
Isopropylbenzene		√	0.4	-	0.008 [H]
Isopropyltoluene, 4-	√	√	0.4	-	-
Methylnaphthalene, 2-	√		0.0031	-	-
Naphthalene	√	√	0.000056	-	0.084 [H]
Propylbenzene, 1-		√	0.15	-	-
Toluene		√	0.4	37	2.14 [H]
Trichloroethene		√	0.00096	-	50 [H]
Trimethylbenzene, 1,2,4-		√	0.0062	-	-
Trimethylbenzene, 1,3,5-	√	√	0.0062	-	-
Xylene, 1,2- [a]		√	0.11	22	0.102 [H]
Xylene, 1,3- and/or 1,4- [a]		√	0.11	22	0.102 [H]
Semivolatile Formulation Components					
Diethylene glycol monoethyl ether	√		0.0031	-	-
Methyl-2-pyrrolidinone, 1-	√		-	-	-
Rotenolone	√	√	-	-	-

- = not available.

PRG =preliminary remediation goal for chronic exposure (USEPA, 2004); based on target HI = 1 or target risk = 1x10-6.

REL =reference exposure level (Cal/EPA, OEHHA, Acute RELs, 2000).

[a] Values apply to mixed xylenes.

[CW] California Water Quality Goals, 8/2003.

Table J-20. Soil Regulatory Values for Rotenone and Other Components of CFT Legumine and NoxFish

Component	Formulation		USEPA Region IX Residential Soil PRG (mg/kg)
	CFT Legumine	NoxFish	
Rotenone	√	√	240
Volatile Formulation Components			
Butylbenzene, 1-	√	√	240
Butylbenzene, sec-	√		220
Isopropylbenzene		√	570
Isopropyltoluene, 4-	√	√	520
Methylnaphthalene, 2-	√		56
Naphthalene	√	√	1.7
Propylbenzene, 1-		√	240
Toluene		√	520
Trichloroethene		√	2.9
Trimethylbenzene, 1,2,4-		√	52
Trimethylbenzene, 1,3,5-	√	√	21
Xylene, 1,2- [a]		√	270
Xylene, 1,3- and/or 1,4- [a]		√	270
Semivolatile Formulation Components			
Diethylene glycol monoethyl ether	√		3,700
Methyl-2-pyrrolidinone, 1-	√		nd
Rotenolone	√	√	nd

nd = not determined.

PRG = preliminary remediation goal for chronic exposure (USEPA, 2004); based on target HI = 1 or target risk = 1x10-6.

[a] Values apply to mixed xylenes.

Table J-21. Projected Ambient Air Exposure Point Concentrations for the Piscicide Formulation Components from Screen3 Modeling

Component	Exposure Point Concentrations in Ambient Air (mg/m³)															
	Proposed Project				Alternative B				Alternative C				Alternative D			
	500 m		1,000 m		500 m		1,000 m		500 m		1,000 m		500 m		1,000 m	
	1-hr max	24-hr avg	1-hr max	24-hr avg	1-hr max	24-hr avg	1-hr max	24-hr avg	1-hr max	24-hr avg	1-hr max	24-hr avg	1-hr max	24-hr avg	1-hr max	24-hr avg
CFT Legumine																
Rotenone	5.49E-03	1.65E-03	3.66E-03	1.10E-03	2.82E-03	8.45E-04	1.51E-03	4.53E-04	7.89E-03	2.37E-03	8.86E-03	2.66E-03	8.99E-03	2.70E-03	1.01E-02	3.03E-03
Butylbenzene, sec-	3.08E-05	9.23E-06	2.05E-05	6.14E-06	1.58E-05	4.73E-06	8.45E-06	2.54E-06	4.42E-05	1.32E-05	4.96E-05	1.49E-05	5.03E-05	1.51E-05	5.65E-05	1.70E-05
Butylbenzene, 1-	6.31E-04	1.89E-04	4.20E-04	1.26E-04	3.23E-04	9.70E-05	1.73E-04	5.20E-05	9.06E-04	2.72E-04	1.02E-03	3.05E-04	1.03E-03	3.10E-04	1.16E-03	3.48E-04
Isopropyltoluene, 4-	2.98E-05	8.95E-06	1.99E-05	5.96E-06	1.53E-05	4.59E-06	8.20E-06	2.46E-06	4.28E-05	1.29E-05	4.81E-05	1.44E-05	4.88E-05	1.46E-05	5.48E-05	1.64E-05
Methylnaphthalene, 2-	7.83E-04	2.35E-04	5.22E-04	1.56E-04	4.02E-04	1.21E-04	2.15E-04	6.46E-05	1.12E-03	3.37E-04	1.26E-03	3.79E-04	1.28E-03	3.84E-04	1.44E-03	4.32E-04
Naphthalene	1.96E-03	5.88E-04	1.30E-03	3.91E-04	1.00E-03	3.01E-04	5.38E-04	1.62E-04	2.81E-03	8.44E-04	3.16E-03	9.48E-04	3.20E-03	9.61E-04	3.60E-03	1.08E-03
Trimethylbenzene, 1,3,5-	3.82E-05	1.14E-05	2.54E-05	7.62E-06	1.96E-05	5.87E-06	1.05E-05	3.15E-06	5.48E-05	1.64E-05	6.15E-05	1.85E-05	6.24E-05	1.87E-05	7.01E-05	2.10E-05
Diethylene glycol monoethyl ether	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Methyl-2-pyrrolidinone, 1-	5.72E-02	1.72E-02	3.81E-02	1.14E-02	2.93E-02	8.80E-03	1.57E-02	4.72E-03	8.22E-02	2.47E-02	9.23E-02	2.77E-02	9.36E-02	2.81E-02	1.05E-01	3.15E-02
Rotenolone	6.74E-04	2.02E-04	4.49E-04	1.35E-04	3.46E-04	1.04E-04	1.85E-04	5.56E-05	9.68E-04	2.90E-04	1.09E-03	3.26E-04	1.10E-03	3.31E-04	1.24E-03	3.72E-04
NoxFish																
Rotenone	6.36E-03	1.91E-03	4.23E-03	1.27E-03	3.26E-03	9.78E-04	1.75E-03	5.24E-04	9.13E-03	2.74E-03	1.03E-02	3.08E-03	1.04E-02	3.12E-03	1.17E-02	3.51E-03
Butylbenzene, 1-	7.10E-02	2.13E-02	4.73E-02	1.42E-02	3.64E-02	1.09E-02	1.95E-02	5.85E-03	1.02E-01	3.06E-02	1.14E-01	3.43E-02	1.16E-01	3.48E-02	1.30E-01	3.91E-02
Isopropylbenzene	5.42E-04	1.63E-04	3.61E-04	1.08E-04	2.78E-04	8.34E-05	1.49E-04	4.47E-05	7.79E-04	2.34E-04	8.75E-04	2.62E-04	8.87E-04	2.66E-04	9.96E-04	2.99E-04
Isopropyltoluene, 4-	7.88E-03	2.37E-03	5.25E-03	1.58E-03	4.04E-03	1.21E-03	2.17E-03	6.50E-04	1.13E-02	3.40E-03	1.27E-02	3.82E-03	1.29E-02	3.87E-03	1.45E-02	4.35E-03
Naphthalene	3.92E-01	1.18E-01	2.61E-01	7.82E-02	2.01E-01	6.03E-02	1.08E-01	3.23E-02	5.62E-01	1.69E-01	6.32E-01	1.90E-01	6.41E-01	1.92E-01	7.20E-01	2.16E-01
Propylbenzene, 1-	1.97E-03	5.91E-04	1.31E-03	3.94E-04	1.01E-03	3.03E-04	5.42E-04	1.63E-04	2.83E-03	8.49E-04	3.18E-03	9.54E-04	3.22E-03	9.67E-04	3.62E-03	1.09E-03
Toluene	1.81E-02	5.43E-03	1.20E-02	3.61E-03	9.27E-03	2.78E-03	4.97E-03	1.49E-03	2.60E-02	7.79E-03	2.92E-02	8.75E-03	2.96E-02	8.88E-03	3.32E-02	9.97E-03
Trichloroethene	7.71E-04	2.31E-04	5.13E-04	1.54E-04	3.95E-04	1.19E-04	2.12E-04	6.35E-05	1.11E-03	3.32E-04	1.24E-03	3.73E-04	1.26E-03	3.78E-04	1.42E-03	4.25E-04
Trimethylbenzene, 1,2,4-	9.54E-02	2.86E-02	6.35E-02	1.91E-02	4.89E-02	1.47E-02	2.62E-02	7.87E-03	1.37E-01	4.11E-02	1.54E-01	4.62E-02	1.56E-01	4.68E-02	1.75E-01	5.26E-02
Trimethylbenzene, 1,3,5-	8.20E-03	2.46E-03	5.46E-03	1.64E-03	4.21E-03	1.26E-03	2.25E-03	6.76E-04	1.18E-02	3.53E-03	1.32E-02	3.97E-03	1.34E-02	4.03E-03	1.51E-02	4.52E-03
Xylene, 1,2-	4.93E-04	1.48E-04	3.28E-04	9.85E-05	2.53E-04	7.58E-05	1.36E-04	4.07E-05	7.08E-04	2.12E-04	7.95E-04	2.39E-04	8.06E-04	2.42E-04	9.06E-04	2.72E-04
Xylene, 1,3- and/or 1,4-	3.96E-03	1.19E-03	2.63E-03	7.90E-04	2.03E-03	6.09E-04	1.09E-03	3.26E-04	5.68E-03	1.70E-03	6.38E-03	1.91E-03	6.47E-03	1.94E-03	7.27E-03	2.18E-03
Rotenolone	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na	na

na = not available.
Bold values are the maximum modeled air concentrations under the Alternative and are selected for comparison to HBSLs.

J.4 EXPOSURE ASSESSMENT

J.4.1 Estimated Exposure Point Concentrations (EPC)

The exposure point concentration (EPC) represents the concentration in the exposure media that would be experienced by humans and/or ecological receptors in the project area. The EPC experienced by a receptor will differ among the media to which they are exposed (i.e., air, water, food, and sediment), by habitat use, by the amount of time spent in the available habitat, and by application rate. For the Proposed Project and alternatives, only one aquatic application rate is proposed (i.e., excluding the no action alternative), and the differences in alternatives relate to the volume of water being treated, not the aquatic concentrations proposed for treatment, as detailed earlier in Table J-1.

J.4.1.1 Surface Water

For the estimation of the EPCs in surface water, full mixing was assumed for each of the constituents that were identified in the laboratory analyses of the neat (undiluted) Noxfish® and CFT Legumine® formulations, and the proposed volume of formulation to be used under the Proposed Project and each alternative. These concentrations were previously provided in Table J-15.

J.4.1.2 Air

- Estimations of air concentrations of rotenone and other formulation constituents that could emit from the reservoir after treatment were required to address inhalation risks. These inhalation -derived doses were conservatively modeled using the Screen3 model developed by the USEPA, assuming complete mixing in the water of the rotenone formulations. The upper end estimates of air concentrations from this modeling are provided in Table J-21. The Screen3 model is considered a valid model by the EPA for projecting short-term air concentrations at distances extending from a source. Numerous conservative assumptions are implicit to the model, however, that may greatly exaggerate the actual air concentrations that would be measurable empirically. For the Proposed Project and treatment alternatives, the Screen3 modeling assumed that:
- rotenone formulation constituents were completely mixed in the reservoir;
- maximal phase separation occurred between the air:water interface (i.e., based on chemical-specific properties, as much as is physically possible is assumed to leave the water and enter the air);
- constituents in the reservoir did not undergo *any* chemical reactions such as hydrolysis or photolysis in the reservoir before volatilization that would essentially reduce their concentration (this overestimates the air concentrations as these natural processes will occur to reduce the chemical concentrations in the air);
- Lake Davis was essentially a rectangular box, with a source height (i.e., point of release of 1 cm (this low height means that there is minimal dilution assumed from the

surrounding air in the source area, and increases the estimated air concentration required to be protective);

- air concentrations are assumed to flow downgradient, with a human receptor height of 1.5 meters (downgradient continuous flow without changing wind direction maximizes the estimated concentration for the downgradient receptor at their breathing height of 1.5 meters); and
- distribution and dilution of all treatment chemicals would be conducted in one 10-hour day, as opposed to two or three days, a time period also captured under the project description in Section 2, and considered much more likely by the DFG due to the logistics inherent to treating the large body of water (the longer distribution period would reduce the maximal concentrations of volatile compounds emitted from the reservoir substantially).

Figure J-5 shows the types of information used in the Screen3 model and the units of those input terms. As part of the conservative approach to this estimation it was also assumed that all of the pesticide needed for each alternative would be applied in one 10-hour day. The project description includes the option to apply the material in the water over 1 to 3 days. It was conservative to assume the shortest time option (a full 10 hour field day) as that assumption maximized the mass of material present in the environment in the shortest time period included in the project description.

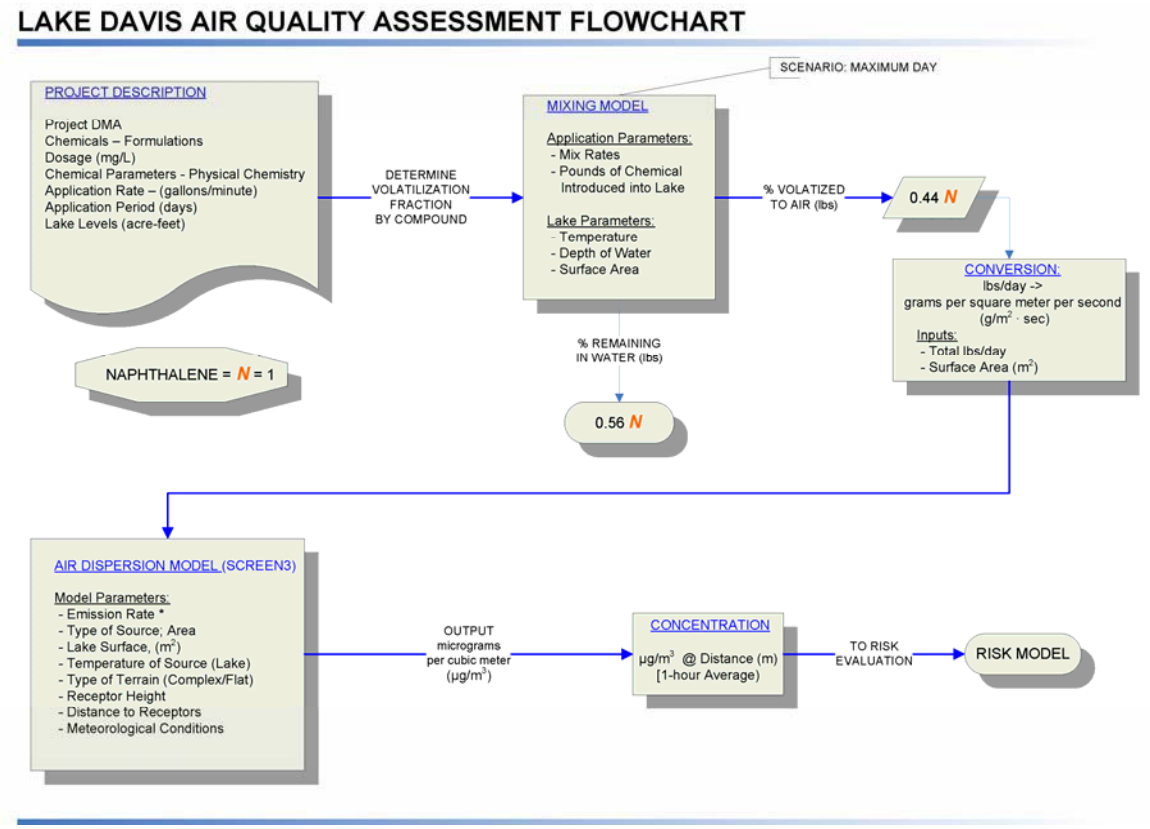


Figure J-5: Lake Davis Air Quality Assessment Flowchart

Figure J-6 provides an example of how the model estimates the concentration gradient in air as the distance increases away from the lake (source area). This example shows the pattern reflected on Table J-21 where the air concentrations are anticipated to peak at a certain distance from the treated water, and then decline steadily with distance. The distance associated with the peak concentration is that distance anticipated to have the longest contact with the volatile compounds in air that could be dispersed away from the treated water by natural movement of the air. (Insert graphic not yet received from Doug) shows a picture of how the model estimates the movement of volatile chemical being emitted from the lake.

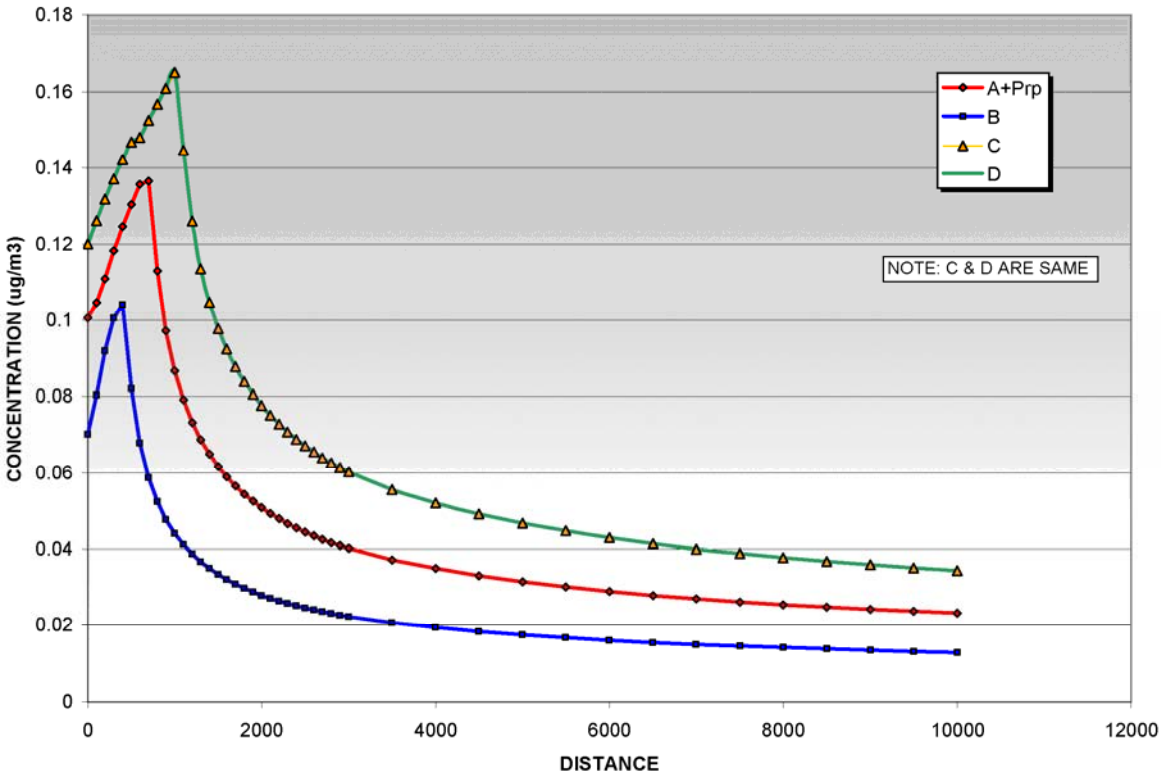


Figure J-6: Air Concentrations Projected from Screen3 Modeling versus Distance from Source

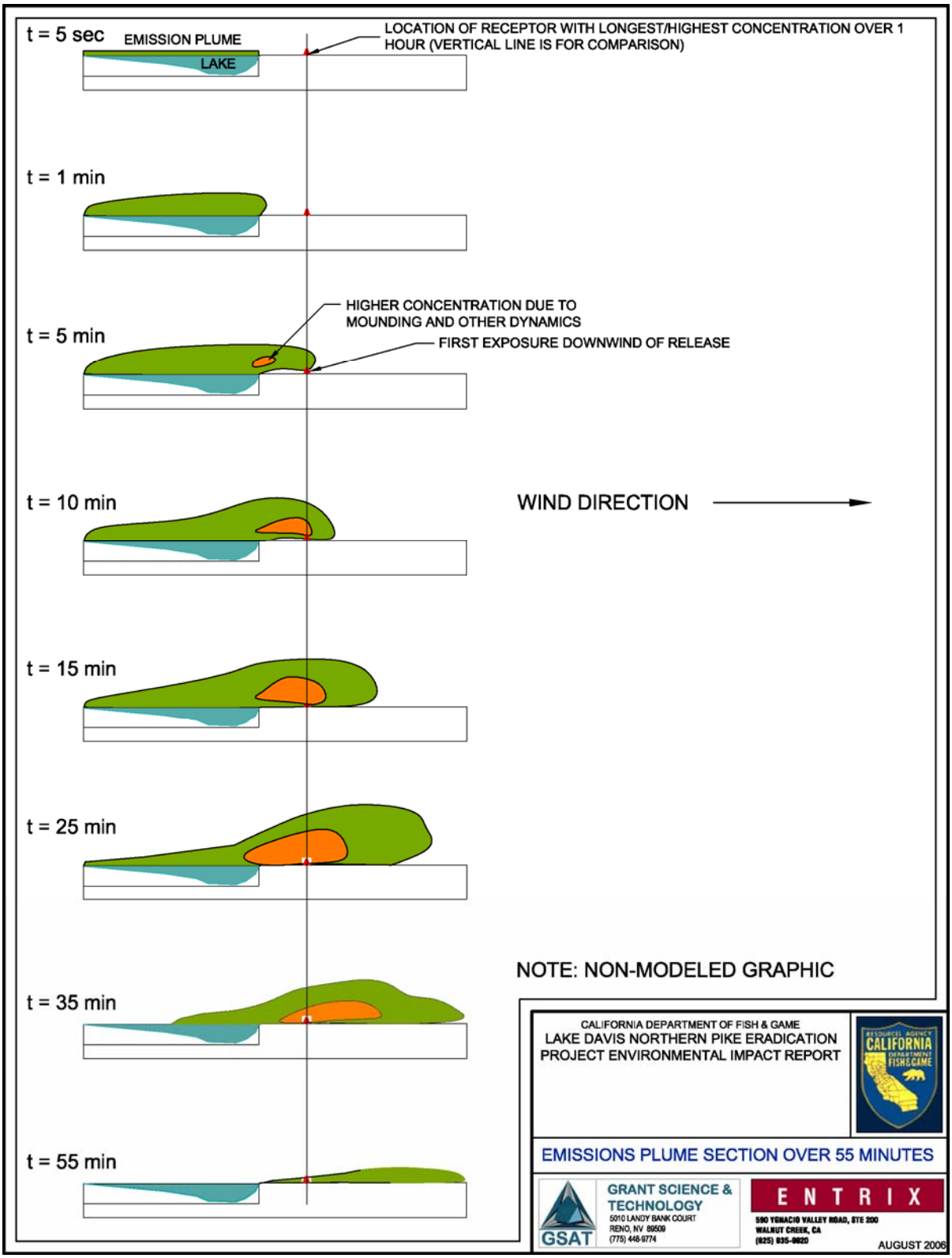


Figure J-7: Emissions Plume Section Over 55 Minutes

Using the Screen3 model as described above, Table J-21 shows the estimated 1 hour maximum air concentration and the 24-hour average air concentration for each formulation constituent for each alternative and the planned applications. The values are shown for 500 and 1000 meters distance downwind from the treatment area. These are the distances that provided the highest modeled air concentrations for the project alternatives and, therefore, offer the most conservative evaluation of potential exposure. There are more sophisticated air modeling techniques available that could provide a more accurate evaluation of potential exposure. However, use of this screening tool is consistent with the level of evaluation in other report sections, and it is unlikely to underestimate potential exposure. The values in bold on the table are those air concentrations selected for use in comparison to the site specific health based screening levels developed in Section 5.0

In interpreting the potential for adverse health concerns, it is important to note that the Screen3 model uses several conservative assumptions in projecting air concentrations from an emission source. As described above under the fate and transport section, all of the formulation constituents naturally and rapidly degrade over time through a variety of physical, chemical and biological mechanisms. In addition, air currents are not likely to be consistently in one direction for the entire potential exposure period (which Screen3 assumes). The pesticide may not all be applied in one 10 hour day (as was assumed) and the potential mixing in the ambient air is likely to be more aggressive than estimated, thereby reducing the potential concentrations. These multiple conservative assumptions represent an upper bound approach, and model, in essence, the a ‘worst case’ scenario for potential air concentrations associated with each project alternative evaluated. Thus, use of the Screen3 model results for subsequent risk assessment likely overestimates potential exposure and associated inhalation risk.

J.4.1.3 Groundwater, Soils and Sediment

Estimates of rotenone formulation constituents in groundwater, soils, and sediments were not modeled from the existing information developed for the Proposed Project and alternatives. Groundwater is not a relevant pathway for exposure to ecological receptors and past monitoring conducted following the previous 1997 treatment of the reservoir; where the same concentration of rotenone was applied as proposed, revealed no detections of rotenone or formulation constituents in community wells that could be attributed to the treatment (DFG 1999). Also see Sections 4.2.4 and 14.2.4.2 of the EIR/EIS.

Soil is also not a complete exposure pathway as indicated in the CSM figures. Areas planned for treatment are not upland soils, and the material is not anticipated to migrate to soils. In addition, if such migration were to occur, the half life of rotenone on exposed soil is very short as described previously.

Sediment concentrations were not modeled for adsorption of formulation constituents from water. In order to have some evaluation of potential sediment concentrations associated with treatment, the data collected following the 1997 application of Nusyn-Noxfish® to Lake Davis were evaluated. The maximum sediment concentrations reported for that treatment program are listed in **Table J-22**. The presence or absence of these chemicals in the undiluted CFT Legumine® and Noxfish® formulations is listed in this table as well for

comparison. The rotenone application rate will be the same; however, some of the dispersant and carrier compounds differ. Significantly, the environmentally persistent piperonyl butoxide that was present as a synergist in the Nusyn-Noxfish® used in 1997 is not present in either of the commercial formulations considered for use, and no other synergist has been added to replace it.

Table J-22. Sediment Exposure Point Concentrations for the Piscicide and Neutralization Formulation Components

Component	Formulation		Concentration in Sediment After Surface Water Treatment with NuSyn-NoxFish[1] (mg/kg)
	CFT Legumine	NoxFish	
Rotenone	√	√	2.10E+00
Butylbenzene, 1-	√	√	na
Butylbenzene, sec-	√		na
Isopropylbenzene		√	na
Isopropyltoluene, 4-	√	√	na
Methylnaphthalene, 2-	√		3.10E-01
Naphthalene	√	√	1.46E-01
Propylbenzene, 1-		√	na
Toluene		√	na
Trichloroethene		√	na
Trimethylbenzene, 1,2,4-		√	na
Trimethylbenzene, 1,3,5-	√	√	na
Xylene, 1,2-		√	na
Xylene, 1,3- and/or 1,4-		√	na
Diethylene glycol monoethyl ether	√		na
Methyl-2-pyrrolidinone, 1-	√		na
Rotenolone	√	√	3.60E-01
Potassium permanganate			na

na = not available.

[1] Siepmann and Finlayson, 1999

J.4.2 Ecological Receptor Exposures

In this section, ecological exposure parameters are provided to estimate the dose of rotenone and the most concentrated formulation constituents for those exposure pathways identified as complete and potentially significant in the conceptual exposure model outlined earlier in Figure J-2. Based on findings reported in Section 7 of the EIR/EIS, a short list of species were selected that could be expected to use the project area for all or a portion of their life history. For the initial screening of exposure and risks, average weights, surface areas, and

daily consumption rates were used to represent exposure. If hazard quotient calculations met or exceeded a level of concern (LOC)(as outlined in Table J-14) then the use of alternative input parameters would be explored. These numbers can exhibit a great deal of variation among populations, but population-specific data from the Lake Davis area were not available.

J.4.2.1 Ecological Receptor Exposure Factors

Exposure factors needed to estimate dose in ecological receptors, such as body weight, food ingestion rate, etc., are summarized in Table J-23 for the list of species for which dose is modeled. These exposure factors were obtained from the Wildlife Exposure Handbook (USEPA 1993), or from Sample et al. (1996). When species-specific data relating to food and water intake were lacking in these compendia references, allometric equations were utilized to estimate the rates of food and/or water ingestion for the receptor species in the same guild.

Allometric equations, used extensively in biological sciences, correlate food and water intake to body weight for free living wild organisms, and are documented in Sample et al. (1996), and "The Wildlife Exposure Factors Handbook" (1993). Separate equations were used for mammals and birds, as documented below.

Food ingestion rate (mammals):

$$Y = 0.235(Wt)^{0.822}$$

Food ingestion rate (birds):

$$Y = 0.648(Wt)^{0.651}$$

Where:

Y = food ingestion rate (g/day)

Wt = representative body weight of a mammalian/avian receptor.

Water ingestion rate (mammals):

$$WI = 0.099(Wt)^{0.90}$$

Water ingestion rate (birds):

$$WI = 0.059(Wt)^{0.67}$$

Where:

WI = water ingestion rate (L/d)

Wt = representative body weight of a mammalian/avian receptor.

Dosage estimates were further developed from the general equation provided in section 2.4 to provide for additional input parameters for select ecological receptors known to use the salt marsh and mud flat areas of Castro Cove using equation [1].

$$[1] \quad \text{Dose} = (\text{SUF}(\text{IR}[\text{food}]*\text{C}[\text{food}]) + (\text{IR}[\text{water}]*\text{C}[\text{water}]) + (\text{IR}[\text{soil}]*\text{C}[\text{soil}]*\text{AE}))/\text{BW}$$

Where:

SUF = Site Use Factor of Habitat Area (percent)

IR = consumption (i.e., intake) rate of [media: food, water, or sediment]

C = concentration of contaminant in [media: food, water, sediment]

AE = assimilation efficiency of contaminants in consumed soil or sediment

BW = Body Weight

For equation [1], the concentration of contaminant in the food was then calculated using equation [2]:

$$[2] \quad \text{Concentration of Contaminant in Food: } \text{C}[\text{food}] = ((\%\ \text{animal matter in diet (BAF)}[\text{animal}]*\text{C}[\text{sed}]) + (\%\ \text{vegetation in diet}(\text{BAF}[\text{veg}]*\text{C}[\text{sed}]))(\text{percent of food contaminated})$$

For this preliminary screening assessment the following conservative exposure assumptions were made:

- BAF was initially assumed to be 1 for both animal matter and vegetable dietary matter.
- SUF was considered 100 percent (i.e., the receptors were contained completely within the project area—a highly conservative assumption for animals with broad home ranges
- Assimilation efficiency of soil or sediment-adsorbed contaminant was 100%
- No additive dose from inhalation was assumed

Table J-23. Exposure Factors for Wildlife and Cattle Used to Assess Risks from Rotenone Use in Lake Davis Project Area

Species	Adult Body Weight (g)	Daily Food Intake (g)	Daily Water Intake (ml)	Inhalation Rate (m^3/day)	Surface Area (cm^2)	Soil & Sediment intake (% of diet)	Relevant Life History Characteristics & Dietary Preference Relevant to Exposure	Conceptual Exposure Pathways
American robin	110	98	1.32	-	-	1	Nesting April-July. Omnivorous: Earthworms, insects, berries.	Primary: Dietary Secondary: Inhalation of drift Tertiary: Water Intake
Bobwhite quail	174	13.5	19	F: 0.10 M: 0.11	F: 298 M: 320	9.3	Breeding in April-July; hatching May to August; Non-migratory; annual mortality rate of approx. 80% Diet: Plants and insects. Max insects 20% in summer	Unlikely for Rotenone application, but considered surrogate for non-water dependent bird species
Marsh wren	11.25	8	3	-	F: 45 M: 48	0	Breed in April; hatch in May; Migration in fall and spring; likely to be found within coastal marsh habitat where *Spartina* is abundant Diet: Insects, spiders, mollusks, and crustaceans.	Primary: Dietary Secondary: Inhalation of drift Tertiary: Water Intake

Table J-23. Exposure Factors for Wildlife and Cattle Used to Assess Risks from Rotenone Use in Lake Davis Project Area

Species	Adult Body Weight (g)	Daily Food Intake (g)	Daily Water Intake (ml)	Inhalation Rate (m^3/day)	Surface Area (cm^2)	Soil & Sediment intake (% of diet)	Relevant Life History Characteristics & Dietary Preference Relevant to Exposure	Conceptual Exposure Pathways
Mallard duck	1,170	420	65	F; 0.42 M: 0.48	F: 1,030 M: 1,148	3.3	A surface feeding "puddle" duck, feeds on an omnivorous diet. Dietary patterns vary with season. In winter, mallards feed mostly on seeds mast, and to a lesser extent invertebrates. In the migratory and breeding seasons, high protein and fat diets are consumed, with more invertebrate biomass.	Primary: dietary exposure through animal, plant and sediment ingestion, and feather preening. Secondary: inhalation of drift Tertiary: water intake
Scaup	770	50	46	F: 0.34 M: 0.36	F: 842 M: 906	3.3	Pacific Flyway spring migration from March—April; fall migration from September-mid-October. Diet: Juveniles ate entirely animal matter in NW territories study; 61% animal matter in Louisiana study,	Primary: Dietary Secondary: drinking water Tertiary: inhalation
Great blue heron	2,230	650	100	-	-	9.4	Diet: Fish, amphibians, snakes & lizards, large insects and small mammals.	Primary: Dietary Secondary: Drinking water Tertiary: Inhalation

Table J-23. Exposure Factors for Wildlife and Cattle Used to Assess Risks from Rotenone Use in Lake Davis Project Area

Species	Adult Body Weight (g)	Daily Food Intake (g)	Daily Water Intake (ml)	Inhalation Rate (m^3/day)	Surface Area (cm^2)	Soil & Sediment intake (% of diet)	Relevant Life History Characteristics & Dietary Preference Relevant to Exposure	Conceptual Exposure Pathways
Bald eagle	3,750	900	160	F: 1.43 M: 1.19*	F: 2,970 M: 2,530*	5.9	Usually associated near large bodies of water. Diet: Fishes, waterfowl, small mammals & carrion.	Primary: Dietary Secondary: Water intake Tertiary: Inhalation
Deer mouse	21	2.8	7	F: .025 M: 023	F: 86 M: 91	2	Breed several times during the year. Diet: Mixture of nuts, seeds, and insects	Primary: ingestion of grain, habitat use limited, however. Secondary; drinking Tertiary: inhalation
Cottontail rabbit	1,200	79	116	0.63	1,254	6.3	Breed several times during the year Diet: Grasses, shrubs, woody plants	Primary: ingestion of treated plant matter Secondary: drinking water Tertiary: inhalation
Norway rat	300	15	33	No data	500	2	Breed several times during the year. Diet: Omnivorous	Primary: dietary Secondary: water intake Tertiary: skin contact (Inhalation exposure unlikely due to nocturnal behavior)
Red Fox	4,530	237	428	F: 1.7 M: 2.0	F: 2760 M: 3220	2.8	Breeding in December – February Diet: Omnivorous: mostly small mammals, birds, insects, and fruit. Plant material is common in summer and fall diet.	Primary: dietary Secondary: water intake Tertiary: skin contact

Table J-23. Exposure Factors for Wildlife and Cattle Used to Assess Risks from Rotenone Use in Lake Davis Project Area

Species	Adult Body Weight (g)	Daily Food Intake (g)	Daily Water Intake (ml)	Inhalation Rate (m^3/day)	Surface Area (cm^2)	Soil & Sediment intake (% of diet)	Relevant Life History Characteristics & Dietary Preference Relevant to Exposure	Conceptual Exposure Pathways
Mule deer	75,470	2400	4,800	M: 30.05* F: 17.26	M: 28,468.5* F: 18,142.4	6.8	Breeding in June. Diet: Herbivorous: leaves & twigs of trees & shrubs. Acorns, legumes & fleshy fruits	Primary: water intake Secondary: inhalation of drift Tertiary: dietary from inadvertently treated vegetation
Black bear	128,870	3900	7,800	M: 67.05* F: 43.19	M: 54,641.8* F:38,220.6	2.8	Hibernation period: 3-4 months during winter (January-April) Diet: Omnivorous: Grasses & Forbes in spring, fruits in summer, nuts & acorns in fall, insects & beetles. Carrion.	Primary: water intake Secondary: dietary—consumption of dead fish Tertiary: inhalation
Cow and calf	599,000	11800	94,600	90.99*	70,021.7*	20 (estimate)	Drink vast quantities of water. Diet: Grasses.	Primary: water intake Secondary: inhalation of drift Tertiary: dietary ingestion of treated vegetation
Pacific Treefrog	2.27	0.0245	No data	No data	1.82*	20 (estimate)	Breeding from January-mid May. Tadpoles 100% aquatic. Diet: Plant material as juveniles; insects as adults.	Primary: Dermal contact across skin membrane Secondary: Inhalation of drift.

Table J-23. Exposure Factors for Wildlife and Cattle Used to Assess Risks from Rotenone Use in Lake Davis Project Area

Species	Adult Body Weight (g)	Daily Food Intake (g)	Daily Water Intake (ml)	Inhalation Rate (m^3/day)	Surface Area (cm^2)	Soil & Sediment intake (% of diet)	Relevant Life History Characteristics & Dietary Preference Relevant to Exposure	Conceptual Exposure Pathways
Western Toad	52	0.5	No data	No data	M: 14.3* F: 17.64	25 (estimate)	Aquatic habitat. Diet: Plant material as juveniles; insects & beetles as adults.	Primary: Dermal contact across skin membrane. Secondary: Inhalation of drift
Long-Toed Salamander	4.6	0.04	No data	No data	M: 29.2* F: 24.3	25 (estimate)	Typically absent from fish holding water bodies. Diet: Juveniles: zooplankton & small macroinverts. Adults: aquatic & terrestrial inverts.	Primary: Dermal contact across skin membrane. Secondary: Inhalation of drift
Pond Turtle	854	19.6	0.35	No data	No data	5.9	Aquatic. Diet: Aquatic macroinvertebrates.	Primary: Dietary Secondary: Inhalation of drift
Common Garter snake	210	5	0.1	No data	No data	5.9	Aquatic. Diet: Amphibians, earthworms & fish.	Primary: Dietary Secondary: Inhalation of drift

*Estimated

J.4.2.2 Mammalian Wildlife Exposures

Mammalian wildlife can be exposed to rotenone and other measured formulation constituents through dermal, oral (ingestion of food and/or water) or inhalation routes. For this assessment, only the ingestion routes (diet, water, and soils/sediment) were considered complete and potentially significant. Dermal exposure was considered either incomplete, or insignificant. We modeled exposure to seven mammalian species: the cottontail rabbit, Norway rat, deer mouse, fox, black bear, and mule deer as representative mammalian wildlife that have been documented in the project area, or that have been the foundation for much of the toxicological effects literature (e.g., rat).

J.4.2.3 Avian Exposure

Exposure for birds may occur via the same pathways as mammals: ingestion, contact, and inhalation. The broad array of life history behaviors among birds prevents an assessment of all bird species that could use the project area. We therefore modeled potential ingestion doses to the bobwhite quail, marsh wren, mallard duck, scaup, great blue heron, bald eagle, and American robin to provide for a range of dietary habits and life history behaviors. Contact was considered a potentially complete pathway, but insignificant due to the feather barrier. All of these species, with the exception of the bobwhite quail, could be found in the project area. The bobwhite quail was included in the modeling because of its common use as a laboratory test species.

J.4.2.4 Aquatic Animal Exposure

Exposure to fish and aquatic invertebrates to rotenone and rotenone formulation constituents from the Proposed Project constitutes a complete pathway and exposure will be significant through bioconcentration. Therefore, we assumed the maximum EPC to correspond to the diluted product at full mixing, as illustrated in the aquatic concentrations identified in Table J-15 (except for rotenone, which was assumed to be fully diluted to 50 ppb, per the project description under each treatment alternative). We then compared the TRVs for the fish and aquatic invertebrate species against the EPC for rotenone. Given the substantially greater sensitivity of aquatic animals to rotenone versus other constituents, it was considered unnecessary to evaluate potential aquatic-derived doses (exposure) from the other formulation constituents, although some potential for transient additivity may be possible.

Estimating exposure to sediment-adsorbed rotenone and other formulation constituents was considered unnecessary as the principal route of exposure will be through bioconcentration from constituents dissolved in surface water.

J.4.2.5 Reptile and Amphibian Exposure

Although dietary uptake of rotenone formulation constituents is possible and was modeled using the methods outlined above (i.e., with a large degree of uncertainty for this class of animals), the most direct exposure pathways for reptiles and amphibians is through dermal contact and/or across the gills (i.e. for juvenile amphibians) when resident in the treated

waters. Additional direct contact exposure with spray administered via backpack is possible to reptiles and amphibians in the riparian and littoral zones of the treated water bodies. However, given that applicators will not be intentionally applying rotenone to riparian and littoral vegetation and soils, and would stop spraying to avoid reptiles and amphibians they may encounter during application, we have assumed this exposure pathway is possible, but likely insignificant. Given that rotenone has been demonstrated to elicit toxicity directly through skin and gill absorption through the water, and that the most sensitive life stages of amphibians are the juvenile stages with gills, we characterized risks to amphibians by comparing aquatic sensitivities to the surface water EPCs.

J.4.3 Human Population Exposure Assessment

The exposure assessment is a critical element risk assessment because this component identifies which potential human exposure pathways and populations are included for further quantitative risk characterization. The key elements of this effort included:

- Identifying relevant exposure pathways associated with the current and future land uses of the site based on the CSM, and
- Identifying and developing relevant exposure factors for each significant subpopulation and pathway to be used in the derivation of health-based screening levels (HBSLs)

Table J-24 below summarizes those pathways depicted with closed circles in the CSM for human health (Figure J-3) for each of the receptors being evaluated. The quantitative evaluation will include calculation of Health Based Screening Levels (HBSLs) that are inclusive of these exposure pathways for each receptor type.

Table J-24. Final List of Complete Exposure Pathways for Which Human Health Risks Will be Evaluated

Exposure Population	Exposure Pathways
Nearby Residents (downwind of Lake Davis)	Inhalation of vapors during direct application of piscicide to surface water, and volatilization from surface water following application activities
Nearby Workers (downwind of Lake Davis)	
Recreational Child Camper	Inhalation of vapors during direct application of piscicide to surface water, and volatilization from surface water following application activities. Incidental ingestion and dermal contact with surface water and sediment.
Unauthorized Youth	

J.4.3.1 Exposure Factors and Calculations to Estimate Human Exposure

There are various equations that are used to estimate potential chemical exposure to different environmental media such as the air, soil or sediment, surface water, and groundwater. Some of the equation input terms are general and apply to all receptors, some are receptor specific, and some are chemical specific. The following subsections summarize the exposure factors used to evaluate human health.

The magnitude of human exposure to COPCs in the rotenone formulations is calculated by determining how much of each constituent actually enters the body (referred to as constituent intake). COPC intakes will be quantified using equations presented in relevant regulatory guidance. Site-specific exposure factors were used whenever possible to ensure that the HBSLs derived from these values address the land use and conditions in the project area. For example, exposure assumptions for the recreational child camper were developed by considering the planned camp activities and ages of potential campers. For the nearby resident and worker, standard assumptions were obtained from the appropriate USEPA and state guidance for such variables as body weight, ingestion and inhalation rates, and some exposure frequencies and durations. The source of the assumption is noted on the tables listing the assumptions.

For all exposure scenarios that were evaluated, Reasonable Maximum Exposure (RME) assumptions were used. The use of RME assumptions is standard risk assessment practice, and provides an estimate of potential exposure that represents the reasonable upper bound of potential contact with the media and compounds of concern for the project and its stated alternatives. The RME assumptions provided in USEPA regulatory guidance documents are based on research published in the scientific literature. In general, a distribution of default or standard exposure parameter values are provided in the regulatory guidance for the 90th and 95th percentile confidence level for factors such as exposure duration, ingestion rates, and total exposed skin surface areas. These default values were applied if they were reasonable and applicable for site-specific conditions; otherwise, site-specific exposure factors were used. The exposure factors used for the human health evaluation are described in the following sections and summarized by exposure media in Tables J-25 to J-32.

J.4.3.1.1 General Exposure Factors

Some of the exposure factors identified for the receptors evaluated in this section are general and apply to each of the different receptor types. CF is a soil units conversion factor of 10^{-6} kg/mg. In addition, averaging time (AT) for carcinogenic exposure is 70 years * 365 days/year, or 25,550 days. For noncarcinogenic exposure, AT is equal to the receptor-dependent exposure duration (ED) * 365 days/year.

J.4.3.1.2 Receptor Specific Exposure Factors

EIR/EIS Figure 14-3 reflects the location of potentially specific sensitive land uses, and Figure 14-4 identifies sensitive populations in and adjacent to the project area.

Nearby Residents

Residents living downwind of the piscicide application area are considered receptors of concern in this risk assessment for volatile organic compounds that may migrate in air from the project area during and following the application of piscicide to the lake. To evaluate potential inhalation exposure, default exposure factors were used for a residential adult and child living downwind from the project site (Table J-25). The default residential scenario assumes that a 70-kilogram adult will inhale 20 m^3/day of air while a 15-kilogram child will

inhale 10 m^3/day of air while present at the residence for 24 hours each day. The project use of piscicide is a short term condition, and both the adult and child are assumed to be exposed for 30 days/year for 1 year based on the assumptions of the fish eradication project design. To evaluate potential carcinogenic effects that may not appear in the short term, the adult and child exposures were evaluated separately over a 70-year average life-time. That is, the carcinogenic response was extrapolated out from the short term (30d) estimate of exposure that was considered conservatively realistic with the Proposed Project.

As discussed in Section 2.5.2.3, post treatment monitoring from the previous 1997 rotenone application in Lake Davis in 1997 indicated that the formulation constituents did not reach groundwater wells used by nearby residents. This is why this is not indicated as a complete exposure pathway for humans in Figure J-3. However, the groundwater will also be monitored following this project application of rotenone. Consequently, this evaluation presents HBSLs for the piscicide formulation constituents assuming potable water use of the groundwater. For those compounds that have available MCL values, those should be used to screen the groundwater data. For compounds that do not have MCLs available, these HBSLs can be used as a way to evaluate the health protection of the data to be collected during groundwater monitoring. Table J-26 presents the exposure assumptions for use of groundwater as potable water. The exposure duration is assumed to be 30 days based on the project treatment plans.

Nearby Commercial Worker

Commercial Workers employed downwind of the piscicide application area are receptors of concern for volatile organic compounds that may migrate in air from the project area during and following the application of piscicide to the reservoir. To evaluate potential inhalation exposure, default exposure factors were used for an adult worker spending 8 to 10 hours at work downwind from the project site. Default exposure factors were used for the nearby commercial worker and are presented in Table J-27 .The exposure factors used for the commercial worker are also the same as for the residential adult which assumes that a 70-kilogram adult will inhale 20 m^3/day of air and be exposed for 30 days/year for 1 year based on the assumptions of the pike eradication project design. Although the resident is present for 24 hours a day, several hours are spent at rest. It was assumed that the worker is active during his time at work. Consequently, the inhalation rate of 20 m^3/day is appropriate and consistent with regulatory guidance for both of these receptors. In addition, since the project application and dissipation of the piscicide components is short term (30 days) the resident and worker are assumed to be exposed for this same duration.

Recreational Child

There is a children's summer camp located adjacent to the Ice Pond on Grizzly Creek. Although the chemical treatment and neutralization compounds are not planned to reach that location downstream, this is in the described project area. Therefore, the child recreational camper is included as a receptor of concern in case of accidental release to this area. The child camper is assumed to be within the age range of 7 to 14 which is consistent with the camp literature found at www.grizzlylodge.com. Due to the extensive water sports activities

available at the camp (e.g. swimming, canoeing, sailing, wind surfing, and fishing) it was assumed that the child camper would be exposed to lake surface water and sediments during play activities in and surrounding the lake. Default exposure factors were used when available and are presented in Table J-28. The physical characteristics assumptions such as body weight and skin surface area were taken from regulatory guidance (USEPA 1997a, USEPA 2004c) and averaged for male and female 50th percentile values for the age range. The child recreational scenario assumes that a 38-kilogram child will inhale 12.6 m^3/day of air, 7 days/year for 1 year, and that they will ingest 200 milligrams of soil/sediment. While swimming for 3 hours a day they will ingest 0.15 L/day of surface water, and have 12,300 cm^2 of skin surface area exposed to surface water during swimming (whole body) and 3,800 cm^2 of skin surface area exposed to sediment (forearms, hand, lower legs, and feet). To evaluate potential carcinogenic effects, the child exposure was averaged over a 70-year lifetime, and the noncarcinogenic effects were averaged over a year (365 days).

Unauthorized Youth

Although the park and area immediately around the Lake will be closed to campers during the application, it is possible that an individual could ignore or be unaware of the closing and could camp along the lake perimeter and not be spotted by DFG personnel. This type of receptor was called unauthorized because the area is technically closed to the public during the time when exposure to piscicide components could be possible. It was assumed that the likely person could be a youth aged 12 to 18. This is a more conservative assumption that using an adult for this scenario. The unauthorized youth exposure scenario assumes contact with lake sediment and surface water while recreating in and near the lake. Default exposure factors were used when available and are presented in Table J-29.

The physical characteristics assumptions such as body weight and skin surface area were taken from regulatory guidance (USEPA 1997a, USEPA 2004c) and averaged for male and female 50th percentile values for the age range. The youth-scenario assumes that a 56-kilogram youth will inhale 14 m^3/day of air, 7 days/year for 1 year and incidentally ingest 100 milligrams of sediment, 0.15 liters of surface water per day [based on an intake rate while swimming of 50 mL/hour (USEPA 1989) and an assumed exposure time of 3 hours/day].

They are assumed to have 16,100 cm^2 of skin surface area exposed to surface water during swimming (whole body) and 5,100 cm^2 of skin surface area exposed to sediment (forearms, hand, lower legs, and feet). In addition, a soil-to-skin adherence factor of 21 mg/cm^2 was used to evaluate dermal exposure to sediment [geometric mean value for children-in-mud (USEPA, 2004c); considered conservative for short-term exposure to lake and stream sediment]. To evaluate potential carcinogenic effects, the youth exposure was averaged over a 70-year lifetime, and noncarcinogenic effects were averaged over a year (365 days).

J.4.3.1.3 Chemical Specific Exposure Factors

The amount of chemical that is absorbed through the skin from contact with soil (ABS) depends on the chemical's characteristics. Table J-31 provides the values used for the piscicide components as established in regulatory guidance (Cal/EPA 1999). Absorption of

compounds through the skin from water or the DA_{event} term found on Table J-30 and Table J-31 is estimated according to the equations and information provided in Table J-33. The values generated from those calculations are presented in Table J-32.

Table J-25. Formulas and Factors for Nearby Residential Exposure to Vapors in Ambient Air

Ambient Air (Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED} \qquad HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
AT_{carc}	Period of time over which exposure is averaged for potential carcinogenic effects	25,550 days	70 years * 365 days/year (Cal/EPA 1999).
$AT_{noncarc}$	Period of time over which exposure is averaged for potential noncarcinogenic effects	365 days	ED (years) * 365 days/year (Cal/EPA 1999).
BW	Body weight	70 kg	Default adult body weight (Cal/EPA 1999).
		15 kg	Default child (age = 1 to 6 years, inclusive) body weight (Cal/EPA 1999).
CSF_{inh}	Inhalation cancer slope factor	-- $(mg/kg/day)^{-1}$	Chemical-specific values (Table J-14).
ED	Exposure duration	1 year	Assumed value based on pike eradication strategy.
EF	Exposure frequency	30 days/year	Assumed value based on pike eradication strategy.
$HBSL_{carc}$	Health-based screening level for potential carcinogenic effects	-- mg/m^3	Chemical-specific values (Table J-36).
$HBSL_{noncarc}$	Health-based screening level for potential noncarcinogenic effects	-- mg/m^3	Chemical-specific values (Table J-36).
IRa	Inhalation rate	20 m^3/day	Default adult inhalation rate (Cal/EPA 1999).
		10 m^3/day	Default child inhalation rate (Cal/EPA 1999).
RfD_{inh}	Subchronic inhalation reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
THQ	Target hazard quotient	1 (unitless)	Default value (USEPA 2004b).
TR	Target cancer risk	1E-06 (unitless)	Default value (USEPA 2004b).

USEPA 2004b – PRGs; Cal/EPA 1999 – PEA

Table J-26. Formulas and Factors for Nearby Residential Exposure to Groundwater

Groundwater (Ingestion and Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
AT_{carc}	Period of time over which exposure is averaged for potential carcinogenic effects	25,550 days	70 years * 365 days/year (USEPA 2004b).
$AT_{noncarc}$	Period of time over which exposure is averaged for potential noncarcinogenic effects	365 days	ED (years) * 365 days/year (USEPA 2004b).
BW	Body weight	70 kg	Default adult body weight (Cal/EPA 1999).
CSF_{inh}	Inhalation cancer slope factor	-- $(mg/kg/day)^{-1}$	Chemical-specific values (Table J-14).
CSF_{oral}	Oral cancer slope factor	-- $(mg/kg/day)^{-1}$	Chemical-specific values (Table J-14).
ED	Exposure duration	1 year	Assumed value based on pike eradication strategy.
EF	Exposure frequency	30 days/year	Assumed value based on pike eradication strategy.
$HBSL_{carc}$	Health-based screening level for potential carcinogenic effects	-- mg/L	Chemical-specific values (Table J-37).
$HBSL_{noncarc}$	Health-based screening level for potential noncarcinogenic effects	-- mg/L	Chemical-specific values (Table J-37).
IRa	Inhalation rate	20 m^3/day	Default adult inhalation rate (Cal/EPA 1999).
IRw	Water ingestion rate	2 L/day	Default adult ingestion rate (USEPA 2004b).
RfD_{inh}	Subchronic inhalation reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
RfD_{oral}	Subchronic oral reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
THQ	Target hazard quotient	1 (unitless)	Default value (USEPA 2004b).
TR	Target cancer risk	1E-06 (unitless)	Default value (USEPA 2004b).
VF_w	Volatilization factor for water	0.5 L/m^3	Default value (USEPA 2004b).

USEPA 2004b – PRGs; USEPA 2004c – RAGS E; USEPA 1997a – EFH; USEPA 1989 – RAGS A

Table J-27. Formulas and Factors for Nearby Commercial Worker Exposure to Vapors in Ambient Air

Ambient Air (Inhalation)			
$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$		$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$	
Parameter		**Value**	**Source / Comment**
AT_{carc}	Period of time over which exposure is averaged for potential carcinogenic effects	25,550 days	70 years * 365 days/year (Cal/EPA 1999).
$AT_{noncarc}$	Period of time over which exposure is averaged for potential noncarcinogenic effects	365 days	ED (years) * 365 days/year (Cal/EPA 1999).
BW	Body weight	70 kg	Default adult body weight (Cal/EPA 1999).
CSF_{inh}	Inhalation cancer slope factor	-- $(mg/kg/day)^{-1}$	Chemical-specific values (Table J-14).
ED	Exposure duration	1 year	Assumed value based on pike eradication strategy.
EF	Exposure frequency	30 days/year	Assumed value based on pike eradication strategy.
$HBSL_{carc}$	Health-based screening level for potential carcinogenic effects	-- mg/m^3	Chemical-specific values (Table J-38).
$HBSL_{noncarc}$	Health-based screening level for potential noncarcinogenic effects	-- mg/m^3	Chemical-specific values (Table J-38).
IRa	Inhalation rate	20 m^3/day	Default worker inhalation rate (Cal/EPA 1992).
RfD_{inh}	Subchronic inhalation reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
THQ	Target hazard quotient	1 (unitless)	Default value (USEPA 2004b).
TR	Target cancer risk	1E-06 (unitless)	Default value (USEPA 2004b).

Table J-28. Formulas and Factors for Recreational Child Camper Exposure to Surface Water, Sediment and Vapors in Ambient Air

Surface Water (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

Sediment (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

Ambient Air (Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
ABS	Dermal absorption factor	-- (unitless)	Chemical-specific values (Table J-30).
AF	Soil-to-skin adherence factor	21 mg/cm^2	Geometric mean value for children-in-mud (USEPA 2004c); considered conservative for short-term exposure to lake and stream sediment.
AT_{carc}	Period of time over which exposure is averaged for potential carcinogenic effects	25,550 days	70 years * 365 days/year (USEPA 2004b).
$AT_{noncarc}$	Period of time over which exposure is averaged for potential noncarcinogenic effects	365 days	ED (years) * 365 days/year (USEPA 2004b).
BW	Body weight	38 kg	Average (male and female) of 50th percentile body weights for age = 7 to 14 years, inclusive (USEPA, 1997a, Tables 7-6 and 7-7).
CF	Units conversion factor for soil/sediment	1E-06 kg/mg	--
CSF_{inh}	Inhalation cancer slope factor	-- (mg/kg/day)$^{-1}$	Chemical-specific values (Table J-14).
CSF_{oral}	Oral cancer slope factor	-- (mg/kg/day)$^{-1}$	Chemical-specific values (Table J-14).
DA_{event}	Dose absorbed per unit area per event	-- L/cm^2/event	Chemical-specific values derived using formulas developed in RAGS E (USEPA 2004c) and reproduced in Table J-31. Values calculated in Table J-32.
ED	Exposure duration	1 year	Assumed value based on pike eradication strategy.
EF	Exposure frequency	14 days/year	2-week session for campers at Walton's Grizzly Lodge.
EV	Event frequency	1 event/day	Assumes that an unauthorized youth will contact surface water once per day.

Table J-28. Formulas and Factors for Recreational Child Camper Exposure to Surface Water, Sediment and Vapors in Ambient Air

Surface Water (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

Sediment (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

Ambient Air (Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
$HBSL_{carc}$	Health-based screening level for potential carcinogenic effects	-- mg/m^3	Chemical-specific values for ambient air (Table J-39).
		-- mg/kg	Chemical-specific values for sediment (Table J-41).
		-- mg/L	Chemical-specific values for surface water (Table J-40).
$HBSL_{noncarc}$	Health-based screening level for potential noncarcinogenic effects	-- mg/m^3	Chemical-specific values for ambient air (Table J-39).
		-- mg/kg	Chemical-specific values for sediment (Table J-41).
		-- mg/L	Chemical-specific values for surface water (Table J-40).
IRa	Inhalation rate	12.6 m^3/day	Average (male and female) of daily inhalation rates for age = 7 to 14 years, inclusive (USEPA 1997a, Table 5-23).
IRs	Soil/sediment ingestion rate	200 mg/day	Default child soil ingestion rate used to represent recreational children aged 7 to 14 years, inclusive (USEPA 2004b).
IRw	Surface water ingestion rate	0.15 L/day	Based on an intake rate while swimming of 50 mL/hour (USEPA 1989) and an assumed exposure time of 3 hours/day.
SA	Skin surface area available for contact	3,800 cm^2/day	Sediment: assumed average (male and female) of 50th percentile surface areas for forearms, hands, lower legs, and feet for age 7 to 14 years, inclusive (USEPA 2004c, Exhibit C-1).
		12,300 cm^2	Surface water: assumed average (male and female) of 50th percentile total body surface areas while swimming for age 7 to 14 years, inclusive (USEPA 2004c, Exhibit C-1).

Table J-28. Formulas and Factors for Recreational Child Camper Exposure to Surface Water, Sediment and Vapors in Ambient Air

Surface Water (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

Sediment (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

Ambient Air (Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
RfD_{inh}	Subchronic inhalation reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
RfD_{oral}	Subchronic oral reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
THQ	Target hazard quotient	1 (unitless)	Default value (USEPA 2004b).
TR	Target cancer risk	1E-06 (unitless)	Default value (USEPA 2004b).

USEPA 2004b – PRGs; USEPA 2004c – RAGS E; USEPA 1997a – EFH; USEPA 1989 – RAGS A

Table J-29. Formulas and Factors for Unauthorized Youth Exposure to Surface Water, Sediment and Vapors in Ambient Air

Surface Water (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

Sediment (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

Ambient Air (Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
ABS	Dermal absorption factor	-- (unitless)	Chemical-specific values (Table J-30).
AF	Soil-to-skin adherence factor	21 mg/cm^2	Geometric mean value for children-in-mud (USEPA 2004c); considered conservative for short-term exposure to lake and stream sediment.
AT_{carc}	Period of time over which exposure is averaged for potential carcinogenic effects	25,550 days	70 years * 365 days/year (USEPA 2004b).
$AT_{noncarc}$	Period of time over which exposure is averaged for potential noncarcinogenic effects	365 days	ED (years) * 365 days/year (USEPA 2004b).
BW	Body weight	56 kg	Average (male and female) of 50th percentile body weights for age = 12 to 18 years, inclusive (USEPA 1997a, Tables 7-6 and 7-7).
CF	Units conversion factor for soil/sediment	1E-06 kg/mg	--
CSF_{inh}	Inhalation cancer slope factor	-- (mg/kg/day)$^{-1}$	Chemical-specific values (Table J-14).
CSF_{oral}	Oral cancer slope factor	-- (mg/kg/day)$^{-1}$	Chemical-specific values (Table J-14).
DA_{event}	Dose absorbed per unit area per event	-- L/cm^2/event	Chemical-specific values derived using formulas developed in RAGS E (USEPA 2004c) and reproduced in Table J-31. Values calculated in Table J-32.
ED	Exposure duration	1 year	Assumed value based on pike eradication strategy.
EF	Exposure frequency	14 days/year	Assumed value based on pike eradication strategy.
EV	Event frequency	1 event/day	Assumes that an unauthorized youth will contact surface water once per day.

Table J-29. Formulas and Factors for Unauthorized Youth Exposure to Surface Water, Sediment and Vapors in Ambient Air

Surface Water (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

Sediment (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

Ambient Air (Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
$HBSL_{carc}$	Health-based screening level for potential carcinogenic effects	-- mg/m^3	Chemical-specific values for ambient air (Table J-42).
		-- mg/kg	Chemical-specific values for sediment (Table J-44).
		-- mg/L	Chemical-specific values for surface water (Table J-43).
$HBSL_{noncarc}$	Health-based screening level for potential noncarcinogenic effects	-- mg/m^3	Chemical-specific values for ambient air (Table J-42).
		-- mg/kg	Chemical-specific values for sediment (Table J-44).
		-- mg/L	Chemical-specific values for surface water (Table J-43).
IRa	Inhalation rate	14 m^3/day	Average (male and female) of daily inhalation rates for age 12 to 18 years, inclusive (USEPA 1997a, Table 5-23).
IRs	Soil/sediment ingestion rate	100 mg/day	Default adult soil ingestion rate used to represent unauthorized youths aged 12 to 18 years, inclusive (USEPA 2004b).
IRw	Surface water ingestion rate	0.15 L/day	Based on an intake rate while swimming of 50 mL/hour (USEPA 1989) and an assumed exposure time of 3 hours/day.
SA	Skin surface area available for contact	5,100 cm^2/day	Sediment: assumed average (male and female) of 50th percentile surface areas for forearms, hands, lower legs, and feet for age 12 to 18 years, inclusive (USEPA 2004c, Exhibit C-1).
		16,100 cm^2	Surface water: assumed average (male and female) of 50th percentile total body surface areas while swimming for age 12 to 18 years, inclusive (USEPA 2004c, Exhibit C-1).

Table J-29. Formulas and Factors for Unauthorized Youth Exposure to Surface Water, Sediment and Vapors in Ambient Air

Surface Water (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{EF * ED * \left[IRw + \left(DA_{event} * EV * SA\right)\right]}$$

Sediment (Ingestion and Direct Contact)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{oral} * CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{oral} * BW * AT_{noncarc}}{CF * EF * ED * \left[IRs + \left(SA * AF * ABS\right)\right]}$$

Ambient Air (Inhalation)

$$HBSL_{carc} = \frac{TR * BW * AT_{carc}}{CSF_{inh} * IRa * EF * ED}$$

$$HBSL_{noncarc} = \frac{THQ * RfD_{inh} * BW * AT_{noncarc}}{IRa * EF * ED}$$

Parameter		Value	Source / Comment
RfD_{inh}	Subchronic inhalation reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
RfD_{oral}	Subchronic oral reference dose	-- mg/kg/day	Chemical-specific values (Table J-14).
THQ	Target hazard quotient	1 (unitless)	Default value (USEPA 2004b).
TR	Target cancer risk	1E-06 (unitless)	Default value (USEPA 2004b).

USEPA 2004b – PRGs; USEPA 2004c – RAGS E; USEPA 1997a – EFH; USEPA 1989 – RAGS A

Table J-30. Skin Absorption (ABS_D) Values for the COPCs

Component	ABS_D* (mg/cm^2)
Rotenone	0.1
Butylbenzene, 1-	0.1
Butylbenzene, sec-	0.1
Isopropylbenzene	0.1
Isopropyltoluene, 4-	0.1
Methylnaphthalene, 2-	0.15
Naphthalene	0.15
Propylbenzene, 1-	0.1
Toluene	0.1
Trichloroethene	0.1
Trimethylbenzene, 1,2,4-	0.1
Trimethylbenzene, 1,3,5-	0.1
Xylene, 1,2-	0.1
Xylene, 1,3- and/or 1,4-	0.1
Diethylene glycol monoethyl ether	0.1
Methyl-2-pyrrolidinone, 1-	0.1
Rotenolone	0.1
Potassium permanganate	0.01

*All ABS values from Cal/EPA, PEA, 1999 (Appendix A, Table 2).

Table J-31. Formulas and Input Parameters for Calculating DA_{event} for Dermal Exposure to Surface Water

DA_{event} for Organics

Step 1: Calculate K_p (cm/hr) using Equation 3.8 (USEPA, 2004c)

$$K_p = 10^{-2.80 + (0.66\ log\ K_{ow}) - (0.0056\ MW)}$$

Where: K_{ow} = chemical-specific octanol-water partition coefficient (unitless).
MW = chemical-specific molecular weight (g/mol).

Step 2: Calculate B (dimensionless) using Equation A.1 (USEPA, 2004c)

$$B = K_p * \frac{\sqrt{MW}}{2.6}$$

Step 3: Calculate Dsc (cm2/hr) using Equation A.3 (USEPA, 2004c)

$$D_{sc} = l_{sc} * 10^{-2.80 - (0.0056\ MW)}$$

Where: l_{sc} = thickness of the stratum corneum = 0.001 cm.

Step 4: Calculate τevent (hour) using Equation A.4 (USEPA, 2004c)

$$\tau_{event} = \frac{l_{sc}^2}{6\ D_{sc}}$$

Step 5: Calculate t* (hour), based on the value of B using Equations A.5 through A.8 (USEPA, 2004c)

$$If\ B \le 0.6,\ then\ t^* = 2.4\ \tau_{event}$$

$$If\ B > 0.6,\ then\ t^* = 6\ \tau_{event}\left(b - \sqrt{b^2 - c^2}\right)$$

Where:

$$b = \frac{2(1+B)^2}{\pi} - c \quad and \quad c = \frac{1+3B+3B^2}{3(1+B)}$$

Step 6: Calculate DA_{event} (L/cm2-event) using Equations 3.2 and 3.3 (USEPA, 2004c) †

$$If\ t_{event} \le t^*, then\ DA_{event} = 2\ FA * K_p * \sqrt{\frac{6\ \tau_{event} * t_{event}}{\pi}} * CF$$

$$If\ t_{event} > t^*, then\ DA_{event} = FA * K_p * \left[\frac{t_{event}}{1+B} + 2\ \tau_{event}\left(\frac{1+3B+3B^2}{(1+B)^2}\right)\right] * CF$$

Where: CF = 0.001 L/cm^3.
FA = chemical-specific fraction absorbed from water (unitless).
t_{event} = 3 hours/event (assumed value for recreational contact with surface water).

† Constituent concentration in water, CW, is removed from the DA_{event} formulas (Equations 3.2 and 3.3, USEPA, 2004c).

Table J-31. Formulas and Input Parameters for Calculating DA_{event} for Dermal Exposure to Surface Water

DA_{event} for Inorganics †			
$DA_{event}\left(L/cm^2-event\right) = K_p * t_{event} * CF$			
Parameter		**Value**	**Source / Comment**
CF	water conversion factor	0.001 L/cm^3	Standard conversion factor.
K_p	permeability coefficient for water	-- cm/hour	Chemical-specific values. If no chemical-specific K_p is available, the default value for water of 0.001 cm/hour is used.
t_{event}	duration of event	3 hours/event	Assumed value for recreational contact with surface water.

† Constituent concentration in water, CW, is removed from the DA_{event} formulas (Equations 3.2 and 3.3, USEPA, 2004c).

Table J-32. Calculation of DA_{event} for Unauthorized Youth and Camper Exposure to Surface Water

Component	DA_{event} (L/cm^2-event)	B (unitless)	τ_{event} (hour)	t* (hour)	FA (unitless)	K_p (cm/hour)	MW (g/mol)	log K_{ow} (unitless)
Rotenone	8.86E-05	3.82E-02	1.69E+01	4.06E+01	0.9	5.0E-03	394	4.10
Butylbenzene, 1-	7.86E-04	9.79E-01	5.92E-01	6.23E-01	1.0	2.2E-01	134	4.38
Butylbenzene, sec-	1.03E-03	1.29E+00	5.92E-01	7.12E-01	1.0	2.9E-01	134	4.57
Isopropylbenzene	2.47E-04	2.91E-01	4.94E-01	1.19E+00	1.0	6.9E-02	120	3.50
Isopropyltoluene, 4-	5.81E-04	7.12E-01	5.92E-01	5.31E-01	1.0	1.6E-01	134	4.16
Methylnaphthalene, 2-	3.54E-04	4.12E-01	6.56E-01	1.57E+00	1.0	9.0E-02	142	3.86
Naphthalene	1.79E-04	2.05E-01	5.48E-01	1.31E+00	1.0	4.7E-02	128	3.36
Propylbenzene, 1-	2.84E-04	3.37E-01	4.94E-01	1.19E+00	1.0	8.0E-02	120	3.60
Toluene	1.07E-04	1.14E-01	3.44E-01	8.27E-01	1.0	3.1E-02	92	2.75
Trichloroethene	4.86E-05	5.28E-02	5.69E-01	1.37E+00	1.0	1.2E-02	131	2.71
Trimethylbenzene, 1,2,4-	3.80E-04	4.63E-01	4.94E-01	1.19E+00	1.0	1.1E-01	120	3.78
Trimethylbenzene, 1,3,5-	5.04E-04	6.32E-01	4.94E-01	4.17E-01	1.0	1.5E-01	120	4.00
Xylene, 1,2-	1.65E-04	1.86E-01	4.13E-01	9.90E-01	1.0	4.7E-02	106	3.13
Xylene, 1,3- and/or 1,4-	1.84E-04	2.10E-01	4.13E-01	9.90E-01	1.0	5.3E-02	106	3.20
Diethylene glycol monoethyl ether	1.05E-06	1.11E-03	5.92E-01	1.42E+00	1.0	2.5E-04	134	-0.08
Methyl-2-pyrrolidinone, 1-	7.13E-07	7.27E-04	3.77E-01	9.05E-01	1.0	1.9E-04	99	-0.54
Rotenolone	na	na	2.13E+01	na	na	na	412	na
Potassium permanganate	6.00E-06	--	--	--	na	2.0E-03	158	na

na = not available.
DA_{event} methodology presented in USEPA, RAGS E, 2004c; formulas and site-specific factors presented in Table J-31.

J.5 RISK CHARACTERIZATION

J.5.1 Ecological Receptor Risk Characterization

J.5.1.1 Wildlife Risks from Ingestion Pathways

Estimated doses from the food web model exposure pathway to rotenone and the most concentrated constituents in the rotenone formulations are provided in Table J-33. Hazard quotients based on applicable toxicity reference values for acute and chronic exposures are provided in Table J-34. With respect to the TRVs used to characterize risks, the chronic NOAELs were always preferred as they assume the most conservative level of chemical and were therefore used when possible. However, due to lack of toxicology data on certain chemicals, other TRVs such as LOAELs and LD_{50} values were sometimes utilized. Dashes "-" that appear in Table J-34 indicate either: the TRV is not available for the chemical-receptor, or that the value is irrelevant because the preferred NOAEL is used as the TRV. The following discussion provides additional characterization for rotenone and each of the most concentrated COPCs in the formulations to the ecological receptors of interest.

Table J-33. Estimated Ingestion Doses of Most Concentrated Rotenone Formulation Constituents from Combined Food, Water and Sediment Intake

		CFT Legume				*Noxfish*			
Class	**Species**	**Rotenone (50ppb)[a]**	**Diethylene Glycol Monoethyl Ether[b]**	**1-Methyl-2-Pyrrolidinone[c]**	**Naphthalene (0.341ppb)**	**Rotenone (50ppb)**	**Naphthalene (68.326ppb)**	**Toluene**	**1,2,4 Trimethylbenzene**
Avian	American robin	0.046	0.529	0.080	0.0003	0.046	0.062	0.002	0.009
	Bobwhite quail	0.009	0.100	0.015	0.0001	0.009	0.012	0.0003	0.002
	Marsh wren	0.049	0.568	0.086	0.0003	0.049	0.067	0.002	0.01
	Mallard duck	0.021	0.248	0.038	0.0001	0.021	0.029	0.0007	0.004
	Scaup	0.006	0.074	0.011	0.00004	0.006	0.009	0.0002	0.001
	Great blue heron	0.018	0.211	0.032	0.0001	0.018	0.025	0.0006	0.004
	Bald eagle	0.015	0.169	0.026	0.0001	0.015	0.020	0.0005	0.003
Mammalian	Deer mouse	0.024	0.275	0.042	0.0002	0.024	0.032	0.0008	0.005
	Cottontail rabbit	0.001	0.112	0.017	0.0001	0.001	0.013	0.0003	0.002
	Norway rat	0.008	0.095	0.014	0.0001	0.008	0.011	0.0003	0.002
	Red fox	0.007	0.086	0.013	0.0001	0.007	0.010	0.0003	0.001
	Mule deer	0.005	0.057	0.009	0.00003	0.005	0.007	0.0002	0.001

Table J-33. Estimated Ingestion Doses of Most Concentrated Rotenone Formulation Constituents from Combined Food, Water and Sediment Intake

Class	Species	*CFT Legume*				*Noxfish*			
		Rotenone (50ppb)[a]	Diethylene Glycol Monoethyl Ether[b]	1-Methyl-2-Pyrrolidinone[c]	Naphthalene (0.341ppb)	Rotenone (50ppb)	Naphthalene (68.326ppb)	Toluene	1,2,4 Trimethylbenzene
	Black bear	0.005	0.053	0.008	0.00003	0.005	0.006	0.0002	0.001
	Cow & calf	0.009	0.106	0.016	0.0001	0.009	0.012	0.0003	0.002
Reptilian	Pond turtle	0.001	0.014	0.002	0.0001	0.001	0.002	0.00004	0.0002
	Common garter snake	0.001	0.015	0.002	0.0001	0.001	0.002	0.00005	0.0003
Amphibian	Pacific treefrog	0.051	0.589	0.089	0.0003	0.051	0.069	0.002	0.01
	Western toad	0.051	0.589	0.089	0.0003	0.051	0.069	0.002	0.01
	Long-toed salamander	0.051	0.588	0.089	0.0003	0.051	0.069	0.002	0.01

All doses noted in parts per million (ppm)

Table J-34. Wildlife Hazard Quotients From Combined Ingestion Exposure Pathways

			CFT Legumine®					Noxfish®					
Class	Species	Tox. test	Rotenone (50ppb)[a] [High / Average]		Diethylene Glycol Monoethyl Ether[b]	1-Methyl-2-Pyrrolidinone[c]	Naphthalene (0.341ppb)[d]	Rotenone (50ppb)[a] [High / Average]		Naphthalene (68.326ppb)[d]	Toluene[e]	1,2,4 Trimethylbenzene[f]	Level of Concern (LOC)
Avian	American robin	NOAEL	0.501	0.501	0.001	0.0001	-	0.501	0.501	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.062	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Bobwhite quail	NOAEL	0.089	0.089	0.0002	0	-	0.089	0.089	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0012	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Marsh wren	NOAEL	0.122	0.122	0.001	0.0001	-	0.122	0.122	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0067	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Mallard duck	NOAEL	0.372	0.372	0.0005	0	-	0.372	0.372	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0029	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Scaup	NOAEL	0.111	0.019	0.0002	0	-	0.111	0.019	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0009	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Great blue heron	NOAEL	0.569	0.084	0.0004	0	-	0.569	0.084	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0025	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Bald eagle	NOAEL	0.455	0.056	0.0003	0	-	0.455	0.056	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.002	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.1
Mammalian	Deer mouse	NOAEL	0.159	0.159	0.0003	0	-	0.159	0.159	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0032	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Cottontail rabbit	NOAEL	0.126	0.121	0.0002	-	-	0.126	0.121	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	-	-	-	1
		LD50	-	-	-	0	-	-	-	0.0001	-	0	0.5
	Norway rat	NOAEL	0.057	0.057	0.0002	0	-	0.057	0.057	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0011	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Red fox	NOAEL	0.094	0.094	0.0002	0	-	0.094	0.094	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.001	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Mule deer	NOAEL	0.060	0.058	0.0001	0	-	0.060	0.058	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0007	-	-	1

Table J-34. Wildlife Hazard Quotients From Combined Ingestion Exposure Pathways

			CFT Legumine®					Noxfish®					
Class	Species	Tox. test	Rotenone (50ppb)[a] [High / Average]		Diethylene Glycol Monoethyl Ether[b]	1-Methyl-2-Pyrrolidinone[c]	Naphthalene (0.341ppb)[d]	Rotenone (50ppb)[a] [High / Average]		Naphthalene (68.326ppb)[d]	Toluene[e]	1,2,4 Trimethylbenzene[f]	Level of Concern (LOC)
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Black bear	NOAEL	0.049	0.013	0.0001	0	-	0.049	0.013	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0006	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Cow & calf	NOAEL	0.056	0.053	0.0002	0	-	0.056	0.053	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0012	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
Reptilian	Pond turtle	NOAEL	-	-	0	0	-	-	-	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0002	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
	Common garter snake	NOAEL	-	-	0	0	-	-	-	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.0002	-	-	1
		LD50	-	-	-	-	-	-	-	-	-	0	0.5
Amphibian	Pacific treefrog	NOAEL	-	-	0.0012	0.0001	-	-	-	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.007	-	-	1
		LD50	0.095	0.095	-	-	-	0.095	0.095	-	-	0	0.5
	Western toad	NOAEL	-	-	0.0012	0.0001	-	-	-	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.007	-	-	1
		LD50	0.094	0.094	-	-	-	0.094	0.094	-	-	0	0.5
	Long-toed salamander	NOAEL	-	-	0.0012	0.0001	-	-	-	-	0	-	1
		LOAEL	-	-	-	-	0	-	-	0.007	-	-	1
		LD50	0.094	0.094	-	-	-	0.094	0.094	-	-	0	0.5

NOAEL: No observable adverse effect level.
LOAEL: Lowest observable adverse effect level.
LD_{50}: The concentration of chemical leading to a 50 percent mortality of the test animals within a given time period.

Footnotes on Toxicity Reference Values (TRVs):
[a]The rotenone NOAEL value for all mammal and bird species was 0.4mg/kg-bw/day. This value represents the lowest NOAEL value available for separate lab-based studies on rats and dogs (USEPA, 1988; U.S. Fish and Wildlife Service, 1980). The rotenone LD50 value for all amphibian species was 0.58mg/kg. This value represents the lowest LD50 value available for lab-based studies on adult and larval amphibians ().
[b]The Diethylene Glycol Monoethyl Ether NOAEL value for all species was 490mg/kg-bw/day. This value represents the lowest NOAEL value available for lab-based studies on rats (see Table J-15)). No reports on studies using different animal classes were available.
[c]The 1-Methyl-2-Pyrrolidinone NOAEL value for the Norway rat was 3000mg/kg-bw/day based on lab rats. The 1-Methyl-2-Pyrrolidinone NOAEL value for all other species was 1000mg/kg-bw/day. This value represents the lowest available NOAEL obtained from lab-based studies on mice (MSDS Number: B&J 0304, 2001).
[d]The Naphthalene LOAEL value for all mammal and bird species was 10mg/kg-bw/day (NTP, 1992). This value represents the lowest TRV value available for lab-based studies on rats. Although a NOAEL value of 100mg/kg-bw/day was available from lab-based mice studies (NTP, 1980) this was only used for mice given that it was greater than the rat LOAEL.
[e]The Toluene NOAEL value for all mammal and bird species was 312mg/kg-bw/day (NTP, 1990). This value represents the only TRV value available and refers to a lab-based rat study.
[f]The 1,2,4 – Trimethylbenzene LD50 value for all mammal and bird species was 5000mg/kg-bw. This represents the acute 24 hour LD50 value for lab-based studies on rats.

J.5.1.1.1 CFT Legumine

Rotenone

Because the only available NOAELs and LOAELs were for rats and dogs, these were applied across the board even to non-mammalian species. Using these NOAELS or LOAELs was more conservative than the appropriate LD_{50} for certain species because it represented a much lower chemical concentration.

Mammal Risk

As discussed, rotenone is considered as being moderately to highly toxic to mammals, based on the EPA criteria outlined previously (for example, an acute oral LD_{50} of < 500 mg/kg-bw, but >10 mg/kg-bw). However, as demonstrated by the Hazard Quotient (HQ) summary Table J-34, none of the exposure doses exceeded a HQ of 1 for any of the mammal species modeled relative to the NOAEL. This result indicates an insignificant risk from the rotenone in the treatment formula to these receptors.

Avian Risk

Rotenone is considered as being slightly to non-toxic to adult birds, based on the EPA criteria outlined previously. However, some studies have demonstrated that rotenone may be moderately toxic to the nestlings of some species (Cutcomp 1943). Nevertheless, as demonstrated by the Hazard Quotient (HQ) summary table, none of the exposure doses exceeded a HQ of 1 for any of the avian species modeled relative to the NOAEL. This result indicates an insignificant risk from the rotenone in the treatment formula to these receptors.

Reptilian Risk

As demonstrated by the Hazard Quotient (HQ) summary table (Table J-34), none of the exposure doses exceeded a HQ of 1 for either of the reptilian species modeled relative to the NOAEL. This result indicates an insignificant risk from the rotenone in the treatment formula to these receptors.

Amphibian Risk

Rotenone is considered as being highly toxic to amphibians, based on EPA criteria. Juvenile amphibians in the lake at the time of application are likely to be killed by the rotenone due to bioconcentration of the toxicant across their skin and gills, whereas adult amphibians, able to leave aquatic environments for some time, may avoid the initial high exposure levels and efficient uptake of the chemical through their permeable skin. Thus, as with the terrestrial receptors, the amphibian species of concern were modeled relative to the NOAEL in order to determine the level of risk posed by the chemical.

Diethylene Glycol Monoethyl Ether (DGME)

As with rotenone, toxicology data for DGME was limited to a few mammalian species. Therefore, the lowest NOAEL value (490 mg/kg-bw/day for mice) was applied to all subjects.

Mammal Risk

DGME is considered as being practically non-toxic to mammals, based on the EPA criteria, with all studies showing mammalian subjects as having LD50s >5,000 mg/kg (IUCLID 2000). Therefore, it comes as no surprise that, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the mammal species modeled relative to the NOAEL. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Avian Risk

No data were available to demonstrate the toxicity of DGME to birds. Using the mammalian NOAEL value, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the avian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Reptilian Risk

No data were available to demonstrate the toxicity of DGME to reptiles. Using the mammalian NOAEL value, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for either of the reptile species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Amphibian Risk

No data were available to demonstrate the toxicity of DGME to amphibians. Using the mammalian NOAEL value, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the amphibian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

1-Methyl-2-Pyrrolidinone

Again, toxicology data for 1-Methyl-2-Pyrrolidinone was limited to a few mammalian species. Therefore, the lowest NOAEL value (1,000 mg/kg-bw/day for mice) was applied to all subjects except the Norway rat as an appropriate NOAEL value of 3,000 mg/kg-bw/day was available for rats.

Mammal Risk

1-Methyl-2-Pyrrolidinone is considered as being slightly toxic to mammals, based on the EPA criteria, with studies showing mammalian subjects as having LD_{50}s <2,000 mg/kg (B&J

0304, 2001). According to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the mammal species modeled relative to the NOAEL. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Avian Risk

No data were available to demonstrate the toxicity of 1-Methyl-2-Pyrrolidinone to birds. Using the NOAEL value for mice, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the avian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Reptilian Risk

No data were available to demonstrate the toxicity of 1-Methyl-2-Pyrrolidinone to reptiles. Using the NOAEL value for mice, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for either of the reptile species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Amphibian Risk

No data were available to demonstrate the toxicity of 1-Methyl-2-Pyrrolidinone to amphibians. Using the NOAEL value for mice, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the amphibian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Naphthalene

Again, toxicology data for Naphthalene was limited to a few mammalian species. Although a NOAEL for mice was available (100 mg/kg-bw/day), this was in fact larger than the LOAEL for rats (10 mg/kg-bw/day). It was therefore more conservative to apply the LOAEL as the TRV to all receptor species including the mice.

Mammal Risk

Naphthalene is considered as being moderately toxic to mammals, based on the EPA criteria, with studies showing mammalian subjects as having LD_{50}s < 501mg/kg. According to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the mammal species modeled relative to the LOAEL. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Avian Risk

No data were available to demonstrate the toxicity of naphthalene to birds. Using the LOAEL value for rats, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the avian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Reptilian Risk

No data were available to demonstrate the toxicity of Naphthalene to reptiles. Using the LOAEL value for rats, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for either of the reptile species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Amphibian Risk

No data were available to demonstrate the toxicity of Naphthalene to amphibians. Using the LOAEL value for rats, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the amphibian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

J.5.1.1.2 Noxfish

Rotenone

Given the concentration of rotenone used under all Noxfish options is the same as that under CFT Lecumine, the risks can be considered equivalent. Please see discussion of rotenone toxicology above.

Naphthalene

Even though the concentration of naphthalene was much greater in the Noxfish formulation, HQs were all safely below 1 for all species modeled. Please see discussion of naphthalene in J.5.113 for more details.

Toluene

Toxicity data for toluene was limited to rats (NOAEL of 312 mg/kg-bw/day). Therefore, this value was used as the TRV for all species modeled.

Mammal Risk

Toluene is considered as being moderately toxic to mammals, based on the EPA criteria listed in Table J-11, with studies showing mammalian subjects as having LD_{50}s < 501 mg/kg (Neurotoxicology. Vol. 2, Pg. 567, 1981). According to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the mammal species modeled relative to the NOAEL. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Bird Risk

No data were available to characterize the toxicity hazards of toluene to birds. Using the NOAEL value for rats, according to the HQ table, none of the exposure doses exceeded a HQ

of 1 for any of the avian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Reptilian Risk

No data were available to characterize the toxicity hazards of toluene to reptiles. Normally, avian data are used to project risk to reptiles and amphibians in such an event. However, since no avian data were identified, the mammalian NOAEL was used, as derived in laboratory rats. Using the NOAEL value for rats, none of the exposure doses exceeded a HQ of 1 for either of the reptile species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Amphibian Risk

No data were available to characterize the toxicity hazards of toluene to amphibians. Using the NOAEL value for rats, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the amphibian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

1,2,4 Trimethylbenzene

Toxicity data for 1,2,4 Trimethylbenzene was limited to a value of acute toxicity to rats (LD_{50} of 5,000 mg/kg). Therefore, this value was used as the ingestion TRV for all species modeled.

Mammal Risk

1,2,4 Trimethylbenzene is considered as being practically non-toxic to mammals, based on the EPA criteria, with studies showing mammalian subjects as having LD_{50}s > 2,000 mg/kg . According to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the mammal species modeled relative to the LD_{50}. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Bird Risk

No data were available to demonstrate the toxicity of 1,2,4 Trimethylbenzene to birds. Using the LD_{50} value for rats, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the avian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Reptilian Risk

No data were available to demonstrate the toxicity of 1,2,4 Trimethylbenzene to reptiles. Using the LD_{50} value for rats, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for either of the reptile species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

Amphibian Risk

No data were available to demonstrate the toxicity of 1,2,4 Trimethylbenzene to amphibians. Using the LD_{50} value for rats, according to the HQ table, none of the exposure doses exceeded a HQ of 1 for any of the amphibian species modeled. This result indicates an insignificant risk from this chemical in the treatment formula to these receptors.

J.5.1.2 Inhalation Risks to Wildlife

Ambient air concentrations were modeled for the Volatile Organic Compounds (VOCs) and semi-Volatile Organic Compounds (semi-VOCs) contained in the rotenone formulations from Screen3, as provided in Table J-21. These conservatively developed air concentrations were compared against inhalation based TRVs developed in laboratory animals—principally rats or dogs (Table J-16). Air concentrations of all chemicals projected from Screen3 were well below (by several orders of magnitude) the available LC_{50} values for these surrogate wildlife receptors, although for several formulation constituents, no inhalation toxicity data were found readily in the literature (e.g., diethylene glycol ethyl ether, *n*-butylbenzene). These data suggest there is insignificant risk to wildlife receptors from the inhalation of volatilized rotenone formulation constituents.

J.5.1.3 Aquatic Ecological Receptor Risks

Table J-35 summarizes aquatic ecological hazard quotient calculations relative to the surface water EPCs identified in Table J-15, and the aquatic TRVs identified in Table J-10, Table J-11 and Table J-12. As anticipated, fish, larval frogs, and salamanders face significant risk, with toxicity thresholds being exceeded up to 20-fold (i.e., HQs > 20). However, at the proposed concentrations, it appears that the majority of aquatic invertebrate guilds will not be exposed to lethal concentrations, with the exception that Cladocerans and other zooplankton will likely be impacted to some degree.

Table J-35. Aquatic Toxicity Hazard Quotients to Rotenone

			Rotenone TRV			
Class	**Species**	**Surrogate Species**	**Test end Point**	**TRV value (µg/L)**	**Hazard Quotient (HQ)**	**Reference**
Amphibian	Pacific treefrog (adult)	Northern leopard frog (adult)	LC_{50} 24h	240	0.208	1
	Pacific treefrog (larvae)	Northern leopard frog (tadpole)	LC_{50} 24h	5	10	2
	Western toad (adult)	Northern leopard frog (adult)	LC_{50} 24h	240	0.208	1
	Western toad (larvae)	Northern leopard frog (tadpole)	LC_{50} 24h	5	10	2
	Long-toed salamander	Tiger salamander (larvae)	LC_{50} 24h	5	10	2
Fish	Northern pike		LC_{50} 24h	2.3	21.74	3
	Rainbow trout		LC_{50} 24h	3.5	14.29	3
	Largemouth bass		LC_{50} 24h	10	5	3
	Pumpkinseed	Green sunfish	LC_{50} 24h	10.9	4.59	3
	Brown bullhead	Black bullhead	LC_{50} 24h	33.3	1.5	3
	Golden shiner	Common carp	LC_{50} 24h	4.2	11.9	3
Macroinvertebrate	Flatworm	*Catenula* sp.	LC_{50} 24h	5100	0.01	4
		Planaria sp.	LC_{50} 24h	<500	<0.1	4
	Annelid worms	Leech	LC_{50} 48 h	<100	<0.5	4
	Copepod	*Cyclops* sp.	LC100 72h	<100	<0.5	4
	Branchiura	*Argulus* sp.	LC_{50} 24h	~25	~2	4
	Cladoceran	*Daphnia pulex*	LC_{50} 24h	27	1.85	4
		D. pulex	LC_{50} 24h	<25	<2	4
		Diaptomus siciloides	LC_{50} 24h	<25	<2	4
	Conchostracan	*Estheria* sp.	LC_{50} 24h	~50	~1	4
	Freshwater prawn	*Palaemonetes kadiakensis*	LC_{50} 24h	5150	0.01	4
	Crayfish	*Cambarus immunis*	LC_{50} 72h	>500	>0.1	4
	Dragonfly naiad	Macromia sp.	LC_{50} 24h	4700	0.011	4

Table J-35. Aquatic Toxicity Hazard Quotients to Rotenone

Class			Rotenone TRV			
	Species	Surrogate Species	Test end Point	TRV value (µg/L)	Hazard Quotient (HQ)	Reference
	Stonefly naiad	*Pteronarcys californica*	LC_{50} 24h	2900	0.017	4
	Backswimmer	*Notoncta* sp.	LC_{50} 24h	3420	0.015	4
		Notonecta sp.	LC_{50} 24h	~100	~0.5	4
	Caddis fly larvae	*Hydropsychye* sp.	LC_{50} 96h	605	0.083	4
	Whirligig	*Gyrinus* sp.	LC_{50} 24h	3550	0.014	4
	Water mite	Hydrachnidae	LC_{50} 96h	~50	~1	4
	Snail	*Physa pomilia*	LC_{50} 24h	6350	0.008	4
		Oxytrema catenaria	LC_{50} 96h	1750	0.029	4
		Lymnaea stagnalis	LC_{50} 96h	>1000	>0.05	4
	Bivalve Mollusc	*Dreissena polymorpha*	LC_{50} 48h	2190	0.023	4
		Obliquaria reflexa	LC_{50} 48h	>1000	>0.05	4
		Elliptio buckleyi	LC_{50} 96h	2950	0.017	4
		Elliptio complanata	LC_{50} 96h	2000	0.025	4
		Corbicula manilensis	LC_{50} 96 h	7500	0.0067	4
	Ostracod	*Cypridopsis* sp.	LC_{50} 24h	490	0.1	4

Table. Hazard Quotient indices estimated for aquatic receptor organisms inhabiting Lake Davis. HQ index based on a rotenone concentration of 50µg/L or 50 parts per billion (ppb).

References: 1. Farringer, 1972; 2. Hamilton, 1941; 3. Marking & Bills, 1972; 4. Various, summarized by Ling, 2003.

J.5.2 Human Receptor Population Risks

Similar to the ecological risk characterization, the human risk characterization process integrates the results of the hazard assessment and toxicity information with the human exposure assessment to evaluate potential risks. This risk characterization was conducted by developing a health-based screening level (HBSL) for each piscicide formulation component for each exposure medium (i.e., surface water, sediment or air) that corresponds to an acceptable risk level (i.e., 1 x 10-6 risk for individual carcinogens and a target hazard index of 1 for individual noncarcinogens).

HBSLs were developed for this project specifically so that comparison values would be available for the short term duration where pesticide components may be present in the environment following the proposed treatment. The chemical specific screening numbers already available in regulatory guidance are based on the assumption that someone contacts that chemical for a long period of time. For example, those values assume that a resident breathes the air with the chemical 24 hours a day, 7 days a week for 30 years, with 6 of those years spent as a child in the same location. Since the fate and transport information available for the pesticide components, as described above, indicate that these compound do not persist in the environment for that extended period of time, and that they break down naturally to harmless components, such a long term exposure is inappropriate for this project. Therefore, the risk assessment process and calculation methods identical to those used in developing the chronic (long-term) screening value were used to develop project specific screening numbers (HBSLs) that reflect concentrations that are safe for continuous contact for approximately 30 days. This time period was selected, since previous monitoring data from the previous application indicated the constituents associated with the proposed pesticide formulations will not be present after that length of time in the environment, since they will have degraded. The specific time period and contact assumptions used to develop the HBSLs are described above in Section J.4.3.1.3

The results of this calculation process are chemical concentrations of the formulation components that are health protective if present in the exposure medium following piscicide application as described for the project. These HBSL concentrations can be compared to predicted or measured concentrations of the formulation constituent in the environment to determine if any mitigation measures are needed for the proposed project to either reduce exposure point concentrations or restrict/eliminate potentially hazardous exposure pathways. The HBSLs produced in this evaluation serve as benchmarks for comparison to concentrations of the same compounds in the environment.

The risk characterization is divided into two subsections. The first describes the general methodology for deriving HBSLs based on carcinogenic and noncarcinogenic effects, and the second is a summary of the risk results for each human receptor group.

J.5.2.1 General Methodology for Deriving HBSLs

The methodology for estimating HBSLs is based on Federal and State guidance documents developed for risk assessment (Cal/EPA, 1992, 1994b; USEPA, 1989, 1991a). Three types of information are needed to calculate HBSLs:

1. Level of human intake associated of each exposure medium as described in the exposure section,
2. The relationship between intake of the chemical and its toxicity as described in the hazard assessment, and
3. Acceptable target cancer risk and noncarcinogenic hazard index (HI) values as a basis for HBSL development.

The target risk and HI values used for the identified human receptor populations for this project are 10^{-6} and 1, respectively. These values are consistent with the levels developed by

USEPA Region 9 (2004) for health-based screening evaluations and levels used in CalEPA guidance (1994b). Potentially complete exposure pathways identified in the CSM (Figure J-4) were used for estimating HBSLs. Pathway specific exposure factors for calculating carcinogenic and noncarcinogenic intake were previously presented in Tables J-25 to J-32. The resulting HBSLs for the project constituents are presented in Tables J-36 to J-44 and are described in the sections below. Tables J-45 to J-50 compare the calculated site specific HBSLs to modeled or surrogate data for formulation constituents.

J.5.2.1.1 HBSLs for Carcinogenic Health Effects

To calculate an HBSL, the risk level is selected and the exposure factors and toxicity information are used to determine the acceptable concentration of a COPC in soil or other relevant media. For lifetime incremental cancer risks, the equation below describes the calculation for determining a chemical-specific HBSL for intake by a given receptor (USEPA, 1991a):

$$HBSL_{cancer} = \frac{Target\ Risk\ Level}{\left[CSF_{oral} * \left(Intake_{oral} + Intake_{dermal}\right)\right] + \left[CSF_{inhal} * Intake_{inhal_{vapor}}\right]}$$

Where:

$HBSL_{cancer}$	=	Health-Based Screening Level for carcinogenic effects (mg/kg)
Target Risk Level	=	Acceptable target cancer risk, one in a million (1 x 10-6) (unitless)
Intake	=	Intake for exposure (e.g., kg sediment/kg body weight-day)
CSF	=	Cancer slope factor (mg chemical/kg body weight-day)-1

When using the equation to calculate a cancer HBSL for a particular receptor, only the exposure factors from the exposure pathways that are potentially complete are used.

J.5.2.1.2 HBSLs for Noncarcinogenic Health Effects

The following equation describes the relationship between estimated intake, toxicity, and the potential for noncarcinogenic health effects (USEPA, 1991a):

$$HBSL_{noncancer} = \frac{Target\ Hazard\ Index}{\left(\frac{Intake_{oral} + Intake_{dermal}}{RfD_{oral}}\right) + \left(\frac{Intake_{inhal_{vapor}}}{RfD_{inhal}}\right)}$$

Where:

$HBSL_{noncancer}$	=	Health-Based Screening Level for noncarcinogenic effects (mg/kg)
Target Hazard Index	=	Acceptable Hazard Index (HI), 1 (unitless)

Intake = Intake exposure (oral, dermal, inhalation of particulates) (e.g., kg soil/kg body weight-day)

RfD = Reference Dose or threshold amount of chemical exposure below which adverse health effects are not expected for pathway specific exposures (oral, dermal, inhalation) (mg chemical/kg body weight-day)

When using the equation to calculate a noncancer HBSL for a particular receptor, only the exposure factors from the exposure pathways that are potentially complete are used.

J.5.2.2 Receptor-specific HBSLs

The following subsections describe the HBSLs derived and their comparison to exposure point concentrations previously presented in Table J-15, Table J-23 and Table J-24.

J.5.2.2.1 Nearby Resident

The CSM for human health (Figure J-3) showed that the only complete exposure pathway for the nearby resident is inhalation of formulation constituents from the air. Table J-36 presents the resulting HBSL values for the nearby resident assuming 30 days of continuous exposure for 24 hours a day. The values in bold are the lowest HBSL and will be used to compare to exposure point concentrations.

Table J-36. Derivation of Health-Based Screening Levels: Nearby Residential Exposure to Vapors in Ambient Air

Component	Formulation		Carcinogenic HBSL for Ambient Air (mg/m^3)		Noncarcinogenic HBSL for Ambient Air (mg/m^3)	
	CFT Legumine	NoxFish	Adult	Child	Adult	Child
Rotenone	√	√	nc	nc	1.7E-01	**7.3E-02**
Butylbenzene, 1-	√	√	nc	nc	4.7E+00	**2.0E+00**
Butylbenzene, sec-	√		nc	nc	4.7E+00	**2.0E+00**
Isopropylbenzene		√	nc	nc	4.7E+01	**2.0E+01**
Isopropyltoluene, 4-	√	√	nc	nc	6.0E+01	**2.6E+01**
Methylnaphthalene, 2-	√		nc	nc	1.7E-01	**7.3E-02**
Naphthalene	√	√	2.5E-02	**1.1E-02**	3.7E-02	1.6E-02
Propylbenzene, 1-		√	nc	nc	4.7E+00	**2.0E+00**
Toluene		√	nc	nc	6.0E+01	**2.6E+01**
Trichloroethene		√	4.3E-01	**1.8E-01**	7.2E+00	3.1E+00
Trimethylbenzene, 1,2,4-		√	nc	nc	2.2E-01	**9.3E-02**
Trimethylbenzene, 1,3,5-	√	√	nc	nc	7.2E-01	**3.1E-01**
Xylene, 1,2-		√	nc	nc	3.7E+00	**1.6E+00**

Table J-36. Derivation of Health-Based Screening Levels: Nearby Residential Exposure to Vapors in Ambient Air

Component	Formulation		Carcinogenic HBSL for Ambient Air (mg/m^3)		Noncarcinogenic HBSL for Ambient Air (mg/m^3)	
	CFT Legumine	NoxFish	Adult	Child	Adult	Child
Xylene, 1,3- and/or 1,4-		√	nc	nc	3.7E+00	**1.6E+00**
Diethylene glycol monoethyl ether	√		nc	nc	3.7E-01	**1.6E-01**
Methyl-2-pyrrolidinone, 1-	√		nc	nc	1.8E+00	**7.8E-01**
Rotenolone	√	√	nc	nc	nd	nd
Potassium permanganate			nc	nc	nd	nd

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14 and Table J-25.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

Table J-37 presents the HBSLs for health protective concentrations for groundwater in the event that it is used as potable. Although this was not determined to be a complete exposure pathway, these site-specific HBSLs for potential sub chronic contact with formulation constituents may be useful for use in comparison to groundwater monitoring data in the future. It should be noted that when a chemical has a drinking water Maximum Contaminant Level (MCL) available, that value takes precedence over any other screening value developed, as shown on Table J-37.

Table J-37. Derivation of Health-Based Screening Levels: Nearby Residential Exposure to Groundwater

Component	Formulation		CA MCL[1] (mg/L)	Carcinogenic HBSL for Groundwater (mg/L)	Noncarcinogenic HBSL for Groundwater (mg/L)
	CFT Legumine	NoxFish		Adult	Adult
Rotenone	√	√	na	nc	**2.8E-01**
Butylbenzene, 1-	√	√	na	nc	**7.8E+00**
Butylbenzene, sec-	√		na	nc	**7.8E+00**
Isopropylbenzene		√	na	nc	**6.0E+01**
Isopropyltoluene, 4-	√	√	na	nc	**8.8E+01**
Methylnaphthalene, 2-	√		na	nc	**2.8E-01**
Naphthalene	√	√	na	**4.1E-02**	7.3E-02
Propylbenzene, 1-		√	na	nc	**7.8E+00**
Toluene		√	**1.5E-01**	nc	8.8E+01
Trichloroethene		√	**5.0E-03**	6.2E-01	1.3E-01

Table J-37. Derivation of Health-Based Screening Levels: Nearby Residential Exposure to Groundwater

Component	Formulation		CA MCL[1] (mg/L)	Carcinogenic HBSL for Groundwater (mg/L)	Noncarcinogenic HBSL for Groundwater (mg/L)
	CFT Legumine	NoxFish		Adult	Adult
Trimethylbenzene, 1,2,4-		√	na	nc	**4.3E-01**
Trimethylbenzene, 1,3,5-	√	√	na	nc	**1.4E+00**
Xylene, 1,2-		√	**1.75E+00**	nc	6.7E+00
Xylene, 1,3- and/or 1,4-		√	**1.75E+00**	nc	6.7E+00
Diethylene glycol monoethyl ether	√		na	nc	**7.3E-01**
Methyl-2-pyrrolidinone, 1-	√		na	nc	**3.1E+00**
Rotenolone	√	√	na	nc	nd
Potassium permanganate			na	nc	**3.2E+00**

nc = noncarcinogenic; nd = not determined.

CA MCL = California Maximum Contaminant Level (22 CCR 64444; 6/2004).

All exposure formulas and factors are presented in Table J-14 and Table J-26.

Bold values are the MCL or lowest of the estimated HBSLs for the receptor (if no MCL is available) and are selected for data comparison.

1 Applicable to long-term groundwater ingestion.

J.5.2.2.2 Nearby Worker

The CSM for human health (Figure J-3) showed that the only complete exposure pathway for the nearby worker is inhalation of formulation constituents from the air. Table J-38 presents the resulting HBSL values for the nearby worker assuming 30 days of exposure for 8 to 10 hours a day every work day. The values in bold are the lowest HBSL and will be used to compare to exposure point concentrations.

Table J-38. Derivation of Health-Based Screening Levels: Nearby Worker Exposure to Vapors in Ambient Air

Component	Formulation		Carcinogenic HBSL for Ambient Air (mg/m^3)	Noncarcinogenic HBSL for Ambient Air (mg/m^3)
	CFT Legumine	NoxFish	Adult	Adult
Rotenone	√	√	nc	**1.7E-01**
Butylbenzene, 1-	√	√	nc	**4.7E+00**

Table J-38. Derivation of Health-Based Screening Levels: Nearby Worker Exposure to Vapors in Ambient Air

Component	Formulation		Carcinogenic HBSL for Ambient Air (mg/m^3)	Noncarcinogenic HBSL for Ambient Air (mg/m^3)
	CFT Legumine	NoxFish	Adult	Adult
Butylbenzene, sec-	√		nc	**4.7E+00**
Isopropylbenzene		√	nc	**4.7E+01**
Isopropyltoluene, 4-	√	√	nc	**6.0E+01**
Methylnaphthalene, 2-	√		nc	**1.7E-01**
Naphthalene	√	√	**2.5E-02**	3.7E-02
Propylbenzene, 1-		√	nc	**4.7E+00**
Toluene		√	nc	**6.0E+01**
Trichloroethene		√	**4.3E-01**	7.2E+00
Trimethylbenzene, 1,2,4-		√	nc	**2.2E-01**
Trimethylbenzene, 1,3,5-	√	√	nc	**7.2E-01**
Xylene, 1,2-		√	nc	**3.7E+00**
Xylene, 1,3- and/or 1,4-		√	nc	**3.7E+00**
Diethylene glycol monoethyl ether	√		nc	**3.7E-01**
Methyl-2-pyrrolidinone, 1-	√		nc	**1.8E+00**
Rotenolone	√	√	nc	nd
Potassium permanganate			nc	nd

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14 and Table J-27.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

J.5.2.2.3 Recreational Child Camper

The CSM for human health (Figure J-3) showed that the complete exposure pathways identified for potential contact by the recreational child camper include ambient air, surface water, and sediment. Table J-39 presents the resulting HBSL values for ambient air contact. Table J-40 presents the resulting HBSL values for surface water contact. . It is important to note that the values in Table J-40 include incidental ingestion of surface water for this receptor during water sports activities such as swimming and boating. Evaluation of this receptor does not assume that the lake provides drinking water for the child camper. Table J-41 presents the resulting HBSL values for sediment contact. The values in bold are the lowest HBSL and will be used to compare to exposure point concentrations

Table J-39. Derivation of Health-Based Screening Levels: Recreational Child Camper Exposure to Vapors in Ambient Air

Component	Formulation		Carcinogenic HBSL for Ambient Air (mg/m^3)	Noncarcinogenic HBSL for Ambient Air (mg/m^3)
	CFT Legumine	NoxFish	Child	Child
Rotenone	√	√	nc	**3.1E-01**
Butylbenzene, 1-	√	√	nc	**8.6E+00**
Butylbenzene, sec-	√		nc	**8.6E+00**
Isopropylbenzene		√	nc	**8.6E+01**
Isopropyltoluene, 4-	√	√	nc	**1.1E+02**
Methylnaphthalene, 2-	√		nc	**3.1E-01**
Naphthalene	√	√	**4.6E-02**	6.8E-02
Propylbenzene, 1-		√	nc	**8.6E+00**
Toluene		√	nc	**1.1E+02**
Trichloroethene		√	**7.9E-01**	1.3E+01
Trimethylbenzene, 1,2,4-		√	nc	**4.0E-01**
Trimethylbenzene, 1,3,5-	√	√	nc	**1.3E+00**
Xylene, 1,2-		√	nc	**6.8E+00**
Xylene, 1,3- and/or 1,4-		√	nc	**6.8E+00**
Diethylene glycol monoethyl ether	√		nc	**6.8E-01**
Methyl-2-pyrrolidinone, 1-	√		nc	**3.4E+00**
Rotenolone	√	√	nc	nd
Potassium permanganate			nc	nd

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14 and Table J-28.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

Table J-40. Derivation of Health-Based Screening Levels: Recreational Child Camper Exposure to Surface Water

Component	Formulation		Carcinogenic HBSL for Surface Water (mg/L)	Noncarcinogenic HBSL for Surface Water (mg/L)
	CFT Legumine	NoxFish	Child	Child
Rotenone	√	√	nc	**3.2E+00**
Butylbenzene, 1-	√	√	nc	**1.1E+01**
Butylbenzene, sec-	√		nc	**8.5E+00**
Isopropylbenzene		√	nc	**1.2E+02**
Isopropyltoluene, 4-	√	√	nc	**1.1E+02**
Methylnaphthalene, 2-	√		nc	**8.8E-01**
Naphthalene	√	√	**2.5E-01**	8.4E+01
Propylbenzene, 1-		√	nc	**3.0E+01**
Toluene		√	nc	**5.4E+02**
Trichloroethene		√	7.1E+00	**4.0E-01**
Trimethylbenzene, 1,2,4-		√	nc	**1.0E+02**
Trimethylbenzene, 1,3,5-	√	√	nc	**7.8E+01**
Xylene, 1,2-		√	nc	**9.1E+01**
Xylene, 1,3- and/or 1,4-		√	nc	**8.2E+01**
Diethylene glycol monoethyl ether	√		nc	**3.6E+03**
Methyl-2-pyrrolidinone, 1-	√		nc	**2.7E+02**
Rotenolone	√	√	nc	nd
Potassium permanganate			nc	**3.3E+01**

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14, Table J-28 and Table J-32.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

Table J-41. Derivation of Health-Based Screening Levels: Recreational Child Camper Exposure to Sediment

Component	Formulation		Carcinogenic HBSL for Sediment (mg/kg)	Noncarcinogenic HBSL for Sediment (mg/kg)
	CFT Legumine	NoxFish	Child	Child
Rotenone	√	√	nc	**4.8E+02**
Butylbenzene, 1-	√	√	nc	**1.3E+04**
Butylbenzene, sec-	√		nc	**1.3E+04**
Isopropylbenzene		√	nc	**4.8E+04**
Isopropyltoluene, 4-	√	√	nc	**9.7E+04**
Methylnaphthalene, 2-	√		nc	**3.3E+02**
Naphthalene	√	√	**4.7E+01**	1.6E+04
Propylbenzene, 1-		√	nc	**1.3E+04**
Toluene		√	nc	**9.7E+04**
Trichloroethene		√	6.5E+02	**3.6E+01**
Trimethylbenzene, 1,2,4-		√	nc	**6.1E+04**
Trimethylbenzene, 1,3,5-	√	√	nc	**6.1E+04**
Xylene, 1,2-		√	nc	**2.4E+04**
Xylene, 1,3- and/or 1,4-		√	nc	**2.4E+04**
Diethylene glycol monoethyl ether	√		nc	**7.3E+04**
Methyl-2-pyrrolidinone, 1-	√		nc	**5.2E+03**
Rotenolone	√	√	nc	nd
Potassium permanganate			nc	**7.4E+03**

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14, Table J-28 and Table J-30.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

J.5.2.2.4 Unauthorized Youth

The CSM for human health (Figure J-3) showed that the complete exposure pathways identified for potential contact by the unauthorized youth include ambient air, surface water, and sediment. Table J-42 presents the resulting HBSL values for ambient air contact. Table J-43 presents the resulting HBSL values for surface water contact. It is important to note that the values in Table J-43 include incidental ingestion of surface water by this receptor during water sports activities such as swimming and boating. Evaluation of this receptor does not assume that the lake provides drinking water for the child camper.

Table J-44 presents the resulting HBSL values for sediment contact. The values in bold are the lowest HBSL and will be used to compare to exposure point concentrations.

Table J-42. Derivation of Health-Based Screening Levels: Unauthorized Youth Exposure to Vapors in Ambient Air

Component	Formulation		Carcinogenic HBSL for Ambient Air (mg/m^3) Adolescent	Noncarcinogenic HBSL for Ambient Air (mg/m^3) Adolescent
	CFT Legumine	NoxFish		
Rotenone	√	√	nc	**4.2E-01**
Butylbenzene, 1-	√	√	nc	**1.1E+01**
Butylbenzene, sec-	√		nc	**1.1E+01**
Isopropylbenzene		√	nc	**1.1E+02**
Isopropyltoluene, 4-	√	√	nc	**1.5E+02**
Methylnaphthalene, 2-	√		nc	**4.2E-01**
Naphthalene	√	√	**6.1E-02**	9.0E-02
Propylbenzene, 1-		√	nc	**1.1E+01**
Toluene		√	nc	**1.5E+02**
Trichloroethene		√	**1.0E+00**	1.8E+01
Trimethylbenzene, 1,2,4-		√	nc	**5.3E-01**
Trimethylbenzene, 1,3,5-	√	√	nc	**1.8E+00**
Xylene, 1,2-		√	nc	**9.0E+00**
Xylene, 1,3- and/or 1,4-		√	nc	**9.0E+00**
Diethylene glycol monoethyl ether	√		nc	**9.0E-01**
Methyl-2-pyrrolidinone, 1-	√		nc	**4.5E+00**
Rotenolone	√	√	nc	nd
Potassium permanganate			nc	nd

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14 and Table J-29.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

Table J-43. Derivation of Health-Based Screening Levels: Unauthorized Youth Exposure to Surface Water

Component	Formulation		Carcinogenic HBSL for Surface Water (mg/L) Adolescent	Noncarcinogenic HBSL for Surface Water (mg/L) Adolescent
	CFT Legumine	NoxFish		
Rotenone	√	√	nc	3.7E+00
Butylbenzene, 1-	√	√	nc	1.3E+01
Butylbenzene, sec-	√		nc	9.6E+00
Isopropylbenzene		√	nc	1.4E+02
Isopropyltoluene, 4-	√	√	nc	1.2E+02
Methylnaphthalene, 2-	√		nc	1.0E+00
Naphthalene	√	√	2.8E-01	9.6E+01
Propylbenzene, 1-		√	nc	3.4E+01
Toluene		√	nc	6.2E+02
Trichloroethene		√	8.4E+00	4.7E-01
Trimethylbenzene, 1,2,4-		√	nc	1.2E+02
Trimethylbenzene, 1,3,5-	√	√	nc	8.8E+01
Xylene, 1,2-		√	nc	1.0E+02
Xylene, 1,3- and/or 1,4-		√	nc	9.4E+01
Diethylene glycol monoethyl ether	√		nc	5.3E+03
Methyl-2-pyrrolidinone, 1-	√		nc	3.9E+02
Rotenolone	√	√	nc	nd
Potassium permanganate			nc	4.4E+01

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14, Table J-29 and Table J-32.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

Table J-44. Derivation of Health-Based Screening Levels: Unauthorized Youth Exposure to Sediment

Component	Formulation		Carcinogenic HBSL for Sediment (mg/kg) Adolescent	Noncarcinogenic HBSL for Sediment (mg/kg) Adolescent
	CFT Legumine	NoxFish		
Rotenone	√	√	nc	5.4E+02
Butylbenzene, 1-	√	√	nc	1.5E+04
Butylbenzene, sec-	√		nc	1.5E+04
Isopropylbenzene		√	nc	5.4E+04
Isopropyltoluene, 4-	√	√	nc	1.1E+05
Methylnaphthalene, 2-	√		nc	3.6E+02
Naphthalene	√	√	5.3E+01	1.8E+04
Propylbenzene, 1-		√	nc	1.5E+04
Toluene		√	nc	1.1E+05
Trichloroethene		√	7.3E+02	4.1E+01
Trimethylbenzene, 1,2,4-		√	nc	6.8E+04
Trimethylbenzene, 1,3,5-	√	√	nc	6.8E+04
Xylene, 1,2-		√	nc	2.7E+04
Xylene, 1,3- and/or 1,4-		√	nc	2.7E+04
Diethylene glycol monoethyl ether	√		nc	8.1E+04
Methyl-2-pyrrolidinone, 1-	√		nc	5.8E+03
Rotenolone	√	√	nc	nd
Potassium permanganate			nc	9.4E+03

nc = noncarcinogenic; nd = not determined.

All exposure formulas and factors are presented in Table J-14, Table J-29 and Table J-30.

Bold values are the lowest estimated HBSL for the receptor and are selected for data comparison.

J.5.2.3 Comparison of Short Term Air Screening Values to Exposure Point Concentrations (EPCs)

The purpose of comparing screening values EPCs is to see if there are any anticipated potential exposures that require mitigation measures or institutional controls to protect the public health. There are two types of screening criteria that are relevant to the short term exposure scenario proposed for this project: the site specific HBSLs and Proposition 65 daily dose levels.

The Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) was enacted as a ballot initiative in November 1986. The proposition was intended by its authors to protect California citizens and the state's drinking water sources from chemicals known to

cause cancer, birth defects, or other reproductive harm, and to inform citizens about exposures to such chemicals. Proposition 65 requires the governor to publish, at least annually, a list of chemicals known to the state to cause cancer or reproductive toxicity. The following chemicals are currently listed under Proposition 65 and are components of one or both of the liquid rotenone formulations: N-methyl pyrrolidinone, naphthalene, toluene, and trichloroethylene (Cal/EPA 2006).

The regulation lists an allowable daily amount (presented in μg/day) that may be contacted for each listed chemical (Cal/EPA 2005). For the carcinogens naphthalene and trichloroethylene, the allowable amounts listed are based on the assumption that daily exposure to the compound occurs continuously over a 70 year lifetime. Since this is a short term project, and exposure is for a short period, less than 30 days, these values are not appropriate for screening for this project.

The Proposition 65 lists allowable lifetime exposures based on a cancer risk level of one in one hundred thousand (1×10^{-5}). The project specific HBSLs presented for screening are based on a risk level of one in a million (1×10^{-6}) which is more protective than that required by Proposition 65 for carcinogens.

Two other formulation constituents, n-methyl pyrrolidone and toluene, are listed as reproductive toxins under Proposition 65. The inhalation value given for each listed compound under Proposition 65 was converted to an air concentration screening value for comparison to the modeled air concentrations. This conversion was done by dividing μg/day by 1000 to convert the amount to mg/day. The resulting value was then divided by the inhalation rate (in m^3/day) for each type of receptor as presented in the regulation (CCR, 2003). These Proposition 65 Air Screening Values are presented in the Table J-45.

Table J-45. Proposition 65 Air Screening Values for Reproductive Toxicants

Listed Compound	Prop 65 Daily Value for Inhalation (mg/day) [1]	Inhalation Rate for Receptor (m^3/day) [2]	Prop 65 Air Screening Concentration (mg/m^3)
N-Methyl Pyrrolidone	3.2		
Nearby Resident		20	1.6E-01
Nearby Worker		10	3.2E-01
Child Camper		15	2.1E-01
Unauthorized Youth		15	2.1E-01
Toluene	13		
Nearby Resident		20	6.5E-01
Nearby Worker		10	1.3E+00
Child Camper		15	8.7E-01
Unauthorized Youth		15	8.7E-01

[1]Concentrations listed in the regulation in μg/day were divided by 1000 to convert to mg/day.

[2]Inhalation rates taken from CCR Title 22 Section 12721(d).

J.5.2.3.1 Air EPCs Compared to Screening Levels

Alternative A – Proposed Project

The modeled air concentrations for both CFT Legumine and Noxfish were below the Proposition 65 air screening concentrations for n-methyl pyrrolidone (in CFT Legumine®) and toluene (in Noxfish®).

Table J-46 incorporates the modeled EPC air concentrations (values from Table J-21) for Alternative A (Proposed Project) with the HBSLs protective of inhalation exposure (bold values from Table J-36, Table J-38, Table J-39 and Table J-42) for all receptors evaluated. Table J-46 shows that the air concentrations of CFT Legumine (upper half of the table) do not show any exceedances of the EPCs over the HBSLs. Therefore, the potential air exposures for the planned application of CFT Legumine does not require mitigation measures to protect the public health for any of the receptors evaluated.

In contrast, the lower half of the table shows that naphthalene exceeds all of the HBSLs for inhalation exposure when the Noxfish formulation is used for Alternative A. Naphthalene has a pungent moth ball-like odor that can be irritating to the eyes and throat for some individuals. However, the odor threshold for naphthalene in air is higher than the HBSL values for all of the receptors, indicating that odor is not a health protective indicator for naphthalene. In addition, the one hour maximum for 1, 2, 4-trimethylbenzene is also exceeded. Therefore, use of Noxfish alone for the treatment program may present a potential concern for the public health for this alternative.

It should be noted that using the Screen3 modeling to estimate air concentrations is conservative as is described above in Section J.4.1.2. It is likely that the predicted concentrations are greater than any actual concentrations that may be experienced in the treatment scenarios. Consequently, actual exposures are likely to be less than predicted, and may be significantly less. This approach is used in order to be sure that the general public and ecological receptors are being adequately evaluated and protected.

Table J-46. Comparison of Piscicide Component Concentrations in Ambient Air to Health-Based Screening Levels and Odor Threshold Concentrations Under Alternative A (Proposed)

Component	Modeled Concentration in Ambient Air at 500 meters (mg/m^3)		Health-Based Screening Levels for Vapors in Ambient Air (mg/m^3)				Odor Threshold (mg/m^3)
	1-Hour Maximum	24-Hour Average	Nearby Resident	Nearby Worker	Child Camper	Unauthorized Youth	
CFT Legumine							
Rotenone	5.49E-03	1.65E-03	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	3.08E-05	9.23E-06	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Butylbenzene, sec-	6.31E-04	1.89E-04	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropyltoluene, 4-	2.98E-05	8.95E-06	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Methylnaphthalene, 2-	7.83E-04	2.35E-04	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Naphthalene	1.96E-03	5.88E-04	1.06E-02	2.48E-02	4.59E-02	6.08E-02	4.40E-01
Trimethylbenzene, 1,3,5-	3.82E-05	1.14E-05	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Diethylene glycol monoethyl ether	na	na	1.57E-01	3.66E-01	6.76E-01	8.97E-01	1.12E+00
Methyl-2-pyrrolidinone, 1-	5.72E-02	1.72E-02	7.85E-01	1.83E+00	3.38E+00	4.48E+00	na
Rotenolone	6.74E-04	2.02E-04	Nd	nd	nd	nd	na
NoxFish							
Rotenone	6.36E-03	1.91E-03	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	7.10E-02	2.13E-02	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropylbenzene	5.42E-04	1.63E-04	2.01E+01	4.68E+01	8.65E+01	1.15E+02	
Isopropyltoluene, 4-	7.88E-03	2.37E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Naphthalene	**3.92E-01**	**1.18E-01**	**1.06E-02**	**2.48E-02**	**4.59E-02**	**6.08E-02**	4.40E-01
Propylbenzene, 1-	1.97E-03	5.91E-04	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Toluene	1.81E-02	5.43E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	8.07E+00
Trichloroethene	7.71E-04	2.31E-04	1.83E-01	4.26E-01	7.86E-01	1.04E+00	2.69E+02
Trimethylbenzene, 1,2,4-	**9.54E-02**	2.86E-02	**9.31E-02**	2.17E-01	4.01E-01	5.32E-01	na
Trimethylbenzene, 1,3,5-	8.20E-03	2.46E-03	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Xylene, 1,2-	4.93E-04	1.48E-04	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Xylene, 1,3- and/or 1,4-	3.96E-03	1.19E-03	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Rotenolone	na	na	nd	nd	nd	nd	na

na = not available; nd = not determined.

Conc = modeled air concentration exceeds one or more calculated HBSLs.

Conc = 1-hour maximum concentration exceeds this HBSL.

Conc = 1-hour maximum and 24-hour average concentrations exceed this HBSL.

Alternative B

The modeled air concentrations for both CFT Legumine and Noxfish were below the Proposition 65 air screening concentrations for n-methyl pyrrolidone (in CFT Legumine®) and toluene (in Noxfish®).

Table J-47 incorporates the modeled EPC air concentrations (values from Table J-21) for Alternative B with the HBSLs protective of inhalation exposure (bold values from Table J-36, Table J-38, Table J-39, and Table J-42) for all receptors evaluated. Table J-47 shows that the air concentrations of CFT Legumine (upper half of the table) do not show any exceedances of the EPCs over the HBSLs. Therefore, the potential air exposures for the planned application of CFT Legumine does not require mitigation measures to protect the public health for any of the receptors evaluated.

In contrast, the lower half of the table shows that naphthalene exceeds all of the HBSLs for inhalation exposure when the Noxfish formulation is used for Alternative B. Naphthalene has a pungent moth ball-like odor that can be irritating to the eyes and throat for some individuals. However, the odor threshold for naphthalene in air is higher than the HBSL values for all of the receptors, indicating that odor is not a health protective indicator for naphthalene. In addition, the one hour maximum for 1, 2, 4-trimethylbenzene is also exceeded. Therefore, use of Noxfish alone for the treatment program may present a potential concern for the public health for this alternative.

It should be noted that using the Screen3 modeling to estimate air concentrations is conservative as is described above in Section J.4.1.2. It is likely that the predicted concentrations are greater than any actual concentrations that may be experienced in the treatment scenarios. Consequently, actual exposures are likely to be less than predicted, and may be significantly less. This approach is used in order to be sure that the general public and ecological receptors are being adequately evaluated and protected.

Table J-47. Comparison of Piscicide Component Concentrations in Ambient Air to Health-Based Screening Levels and Odor Threshold Concentrations Under Alternative B

Component	Modeled Concentration in Ambient Air at 500 meters (mg/m^3)		Health-Based Screening Levels for Vapors in Ambient Air (mg/m^3)				Odor Threshold (mg/m^3)
	1-Hour Maximum	24-Hour Average	Nearby Resident	Nearby Worker	Child Camper	Unauthorized Youth	
CFT Legumine							
Rotenone	2.82E-03	8.45E-04	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	1.58E-05	4.73E-06	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Butylbenzene, sec-	3.23E-04	9.70E-05	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropyltoluene, 4-	1.53E-05	4.59E-06	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Methylnaphthalene, 2-	4.02E-04	1.21E-04	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Naphthalene	1.00E-03	3.01E-04	1.06E-02	2.48E-02	4.59E-02	6.08E-02	4.40E-01
Trimethylbenzene, 1,3,5-	1.96E-05	5.87E-06	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Diethylene glycol monoethyl ether	na	na	1.57E-01	3.66E-01	6.76E-01	8.97E-01	1.12E+00
Methyl-2-pyrrolidinone, 1-	2.93E-02	8.80E-03	7.85E-01	1.83E+00	3.38E+00	4.48E+00	na
Rotenolone	3.46E-04	1.04E-04	nd	nd	nd	nd	na
NoxFish							
Rotenone	3.26E-03	9.78E-04	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	3.64E-02	1.09E-02	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropylbenzene	2.78E-04	8.34E-05	2.01E+01	4.68E+01	8.65E+01	1.15E+02	
Isopropyltoluene, 4-	4.04E-03	1.21E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Naphthalene	**2.01E-01**	**6.03E-02**	**1.06E-02**	**2.48E-02**	**4.59E-02**	**6.08E-02**	4.40E-01
Propylbenzene, 1-	1.01E-03	3.03E-04	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Toluene	9.27E-03	2.78E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	8.07E+00
Trichloroethene	3.95E-04	1.19E-04	1.83E-01	4.26E-01	7.86E-01	1.04E+00	2.69E+02
Trimethylbenzene, 1,2,4-	4.89E-02	1.47E-02	9.31E-02	2.17E-01	4.01E-01	5.32E-01	na
Trimethylbenzene, 1,3,5-	4.21E-03	1.26E-03	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Xylene, 1,2-	2.53E-04	7.58E-05	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Xylene, 1,3- and/or 1,4-	2.03E-03	6.09E-04	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Rotenolone	na	na	nd	nd	nd	nd	na

na = not available; nd = not determined.

Conc = modeled air concentration exceeds one or more calculated HBSLs.

Conc = 1-hour maximum concentration exceeds this HBSL.

Conc = 1-hour maximum and 24-hour average concentrations exceed this HBSL.

Alternative C

The modeled air concentrations for both CFT Legumine and Noxfish were below the Proposition 65 air screening concentrations for n-methyl pyrrolidone (in CFT Legumine®) and toluene (in Noxfish®).

Table J-48 incorporates the modeled EPC air concentrations (values from Table J-21) for Alternative C with the HBSLs protective of inhalation exposure (bold values from Table J-36, Table J-38, Table J-39, and Table J-42) for all receptors evaluated. Table J-48 shows that that the air concentrations of CFT Legumine (upper half of the table) do not show any exceedances of the EPCs over the HBSLs. Therefore, the potential air exposures for the planned application of CFT Legumine does not require mitigation measures to protect the public health for any of the receptors evaluated.

In contrast, the lower half of the table shows that naphthalene exceeds all of the HBSLs for inhalation exposure when the Noxfish formulation is used for Alternative C. Naphthalene has a pungent moth ball-like odor that can be irritating to the eyes and throat for some individuals. However, the odor threshold for naphthalene in air is higher than the HBSL values for all of the receptors, indicating that odor is not a health protective indicator for naphthalene. In addition, the one hour maximum for 1, 2, 4-trimethylbenzene is also exceeded. Therefore, use of Noxfish alone for the treatment program may present a potential concern for the public health for this alternative.

It should be noted that using the Screen3 modeling to estimate air concentrations is conservative as is described above in Section J.4.1.2. It is likely that the predicted concentrations are greater than any actual concentrations that may be experienced in the treatment scenarios. Consequently, actual exposures are likely to be less than predicted, and may be significantly less. This approach is used in order to be sure that the general public and ecological receptors are being adequately evaluated and protected.

Table J-48. Comparison of Piscicide Component Concentrations in Ambient Air to Health-Based Screening Levels and Odor Threshold Concentrations Under Alternative C

Component	Modeled Concentration in Ambient Air at 1,000 meters (mg/m^3)		Health-Based Screening Levels for Vapors in Ambient Air (mg/m^3)				Odor Threshold (mg/m^3)
	1-Hour Maximum	24-Hour Average	Nearby Resident	Nearby Worker	Child Camper	Unauthorized Youth	
CFT Legumine							
Rotenone	8.86E-03	2.66E-03	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	4.96E-05	1.49E-05	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Butylbenzene, sec-	1.02E-03	3.05E-04	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropyltoluene, 4-	4.81E-05	1.44E-05	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Methylnaphthalene, 2-	1.26E-03	3.79E-04	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Naphthalene	3.16E-03	9.48E-04	1.06E-02	2.48E-02	4.59E-02	6.08E-02	4.40E-01
Trimethylbenzene, 1,3,5-	6.15E-05	1.85E-05	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Diethylene glycol monoethyl ether	na	na	1.57E-01	3.66E-01	6.76E-01	8.97E-01	1.12E+00
Methyl-2-pyrrolidinone, 1-	9.23E-02	2.77E-02	7.85E-01	1.83E+00	3.38E+00	4.48E+00	na
Rotenolone	1.09E-03	3.26E-04	nd	nd	nd	nd	na
NoxFish							
Rotenone	1.03E-02	3.08E-03	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	1.14E-01	3.43E-02	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropylbenzene	8.75E-04	2.62E-04	2.01E+01	4.68E+01	8.65E+01	1.15E+02	
Isopropyltoluene, 4-	1.27E-02	3.82E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Naphthalene	**6.32E-01**	**1.90E-01**	**1.06E-02**	**2.48E-02**	**4.59E-02**	**6.08E-02**	**4.40E-01**
Propylbenzene, 1-	3.18E-03	9.54E-04	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Toluene	2.92E-02	8.75E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	8.07E+00
Trichloroethene	1.24E-03	3.73E-04	1.83E-01	4.26E-01	7.86E-01	1.04E+00	2.69E+02
Trimethylbenzene, 1,2,4-	**1.54E-01**	4.62E-02	**9.31E-02**	2.17E-01	4.01E-01	5.32E-01	na
Trimethylbenzene, 1,3,5-	1.32E-02	3.97E-03	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Xylene, 1,2-	7.95E-04	2.39E-04	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Xylene, 1,3- and/or 1,4-	6.38E-03	1.91E-03	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Rotenolone	na	na	nd	nd	nd	nd	na

na = not available; nd = not determined.

Conc = modeled air concentration exceeds one or more calculated HBSLs.

Conc = 1-hour maximum concentration exceeds this HBSL.

Conc = 1-hour maximum and 24-hour average concentrations exceed this HBSL.

Alternative D

The modeled air concentrations for both CFT Legumine and Noxfish were below the Proposition 65 air screening concentrations for n-methyl pyrrolidone (in CFT Legumine®) and toluene (in Noxfish®).

Table J-49 incorporates the EPC air concentrations (values from Table J-21) for Alternative D with the HBSLs protective of inhalation exposure (bold values from Table J-36, Table J-38, Table J-39, and Table J-42) for all receptors evaluated. Table J-49 shows that the air concentrations of CFT Legumine (upper half of the table) do not show any exceedances of the EPCs over the HBSLs. Therefore, the potential air exposures for the planned application of CFT Legumine does not require mitigation measures to protect the public health for any of the receptors evaluated.

In contrast, the lower half of the table shows that naphthalene exceeds all of the HBSLs for inhalation exposure when the Noxfish formulation is used for Alternative D. Naphthalene has a pungent moth ball-like odor that can be irritating to the eyes and throat for some individuals. However, the odor threshold for naphthalene in air is higher than the HBSL values for all of the receptors, indicating that odor is not a health protective indicator for naphthalene. In addition, the one hour maximum for 1, 2, 4-trimethylbenzene is also exceeded. Therefore, use of Noxfish alone for the treatment program may present a potential concern for the public health for this alternative.

It should be noted that using the Screen3 modeling to estimate air concentrations is conservative as is described above in Section J.4.1.2. It is likely that the predicted concentrations are greater than any actual concentrations that may be experienced in the treatment scenarios. Consequently, actual exposures are likely to be less than predicted, and may be significantly less. This approach is used in order to be sure that the general public and ecological receptors are being adequately evaluated and protected.

Table J-49. Comparison of Piscicide Component Concentrations in Ambient Air to Health-Based Screening Levels and Odor Threshold Concentrations Under Alternative D

Component	Modeled Concentration in Ambient Air at 1,000 meters (mg/m^3)		Health-Based Screening Levels for Vapors in Ambient Air (mg/m^3)				Odor Threshold (mg/m^3)
	1-Hour Maximum	24-Hour Average	Nearby Resident	Nearby Worker	Child Camper	Unauthorized Youth	
CFT Legumine							
Rotenone	1.01E-02	3.03E-03	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	5.65E-05	1.70E-05	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Butylbenzene, sec-	1.16E-03	3.48E-04	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropyltoluene, 4-	5.48E-05	1.64E-05	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Methylnaphthalene, 2-	1.44E-03	4.32E-04	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Naphthalene	3.60E-03	1.08E-03	1.06E-02	2.48E-02	4.59E-02	6.08E-02	4.40E-01
Trimethylbenzene, 1,3,5-	7.01E-05	2.10E-05	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Diethylene glycol monoethyl ether	na	na	1.57E-01	3.66E-01	6.76E-01	8.97E-01	1.12E+00
Methyl-2-pyrrolidinone, 1-	1.05E-01	3.15E-02	7.85E-01	1.83E+00	3.38E+00	4.48E+00	na
Rotenolone	1.24E-03	3.72E-04	nd	nd	nd	nd	na
NoxFish							
Rotenone	1.17E-02	3.51E-03	7.30E-02	1.70E-01	3.15E-01	4.17E-01	na
Butylbenzene, 1-	1.30E-01	3.91E-02	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Isopropylbenzene	9.96E-04	2.99E-04	2.01E+01	4.68E+01	8.65E+01	1.15E+02	
Isopropyltoluene, 4-	1.45E-02	4.35E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	na
Naphthalene	**7.20E-01**	**2.16E-01**	**1.06E-02**	**2.48E-02**	**4.59E-02**	**6.08E-02**	**4.40E-01**
Propylbenzene, 1-	3.62E-03	1.09E-03	2.01E+00	4.68E+00	8.65E+00	1.15E+01	na
Toluene	3.32E-02	9.97E-03	2.56E+01	5.96E+01	1.10E+02	1.46E+02	8.07E+00
Trichloroethene	1.42E-03	4.25E-04	1.83E-01	4.26E-01	7.86E-01	1.04E+00	2.69E+02
Trimethylbenzene, 1,2,4-	**1.75E-01**	5.26E-02	**9.31E-02**	2.17E-01	4.01E-01	5.32E-01	na
Trimethylbenzene, 1,3,5-	1.51E-02	4.52E-03	3.10E-01	7.24E-01	1.34E+00	1.77E+00	na
Xylene, 1,2-	9.06E-04	2.72E-04	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Xylene, 1,3- and/or 1,4-	7.27E-03	2.18E-03	1.57E+00	3.66E+00	6.76E+00	8.97E+00	4.43E-01
Rotenolone	na	na	nd	nd	nd	nd	na

na = not available; nd = not determined.

Conc = modeled air concentration exceeds one or more calculated HBSLs.

Conc = 1-hour maximum concentration exceeds this HBSL.

Conc = 1-hour maximum and 24-hour average concentrations exceed this HBSL.

J.5.2.3.2 Summary of Air Exceedances

All of the alternatives evaluated for the use of Noxfish showed modeled air concentrations for naphthalene to exceed the HBSL values for various receptors considered. In addition, trimethylbenzene also showed modeled air concentrations exceeding for some alternatives. Table J-50 presents a summary of the modeled air concentrations for these two components of the Noxfish formulation evaluated for this project. The table shows modeled concentrations at increasing distances away from the treatment area, and which receptors have their HBSL exceeded, and if the odor threshold in air is exceeded.

Table J-50 Modeled Air Concentrations for Noxfish That Exceed HBSLs and/or Odor Thresholds

Component/ Distance from Source	Alternative A (Proposed)		Alternative B		Alternative C		Alternative D	
	1-Hour Maximum	24-Hour Average	1-Hour Maximum	24-Hour Average	1-Hour Maximum	24-Hour Average	1-Hour Maximum	24-Hour Average
Naphthalene								
1 m	0.30 C,R,U,W	0.091 C,R,U,W	0.17 C,R,U,W	0.052 C,R,W	0.46 C,R,U,W, O	0.14 C,R,U,W	0.52 C,R,U,W, O	0.16 C,R,U,W
100 m	0.31 C,R,U,W	0.094 C,R,U,W	0.20 C,R,U,W	0.059 C,R,W	0.48 C,R,U,W, O	0.14 C,R,U,W	0.55 C,R,U,W, O	0.16 C,R,U,W
500 m	**0.39 C,R,U,W**	**0.12 C,R,U,W**	**0.20 C,R,U,W**	**0.060 C,R,W**	0.56 C,R,U,W, O	0.17 C,R,U,W	0.64 C,R,U,W, O	0.19 C,R,U,W
1,000 m	0.26 C,R,U,W	0.078 C,R,U,W	0.11 C,R,U,W	0.032 R,W	**0.63 C,R,U,W, O**	**0.19 C,R,U,W**	**0.72 C,R,U,W, O**	**0.22 C,R,U,W**
2,000 m	0.15 C,R,U,W	0.046 C,R,W	0.12 C,R,U,W	0.037 R,W	0.30 C,R,U,W	0.089 C,R,U,W	0.34 C,R,U,W	0.10 C,R,U,W
5,000 m	0.094 C,R,T,W	0.028 R,W	0.043 R,W	0.013 R	0.18 C,R,T,W	0.054 C,R,W	0.20 C,R,T,W	0.061 C,R,T,W
10,000 m	0.070 C,R,U,W	0.021 R	0.032 R,W	-	0.13 C,R,U,W	0.040 R,W	0.15 C,R,U,W	0.045 R,W
1,2,4-Trimethylbenzene								
1 m	-	-	-	-	0.11 R	-	0.13 R	-
100 m	-	-	-	-	0.12 R	-	0.13 R	-
500 m	**0.095 R**	-	-	-	0.14 R	-	0.16 R	-
1,000 m	-	-	-	-	**0.15 R**	-	**0.18 R**	-
2,000 m	-	-	-	-	-	-	-	-
5,000 m	-	-	-	-	-	-	-	-
10,000 m	-	-	-	-	-	-	-	-

Screening Level Type	Screening Level	
	Naphthalene	1,2,4-Trimethylbenzene
C = child camper HBSL	0.046	0.40
R = nearby resident HBSL	0.011	0.093
U =Unauthorized Youth HBSL	0.061	0.53
W = nearby worker HBSL	0.025	0.22
O = odor threshold	0.44	not available

All units are mg/m^3.
Bold value is maximum modeled concentration for the alternative.

J.5.2.3.3 Surface Water EPCs Compared to HBSLs

Table J-51 incorporates the modeled surface water concentrations (values from Table J-15) with the HBSLs protective of surface water contact for swimming and other activities for the child camper and unauthorized youth. This table shows that the surface water EPC concentrations of both CFT Legumine and Noxfish formulation constituents are below the HBSLs. The taste and odor threshold for diethylene glycol monomethly ether in water is exceeded by the one hour maximum concentration for CFT Legumine. However, odor is considered a nuisance endpoint rather than a health concern. The odor of this compound is a sweet ether-like scent that is not typically considered objectionable. Therefore, the potential surface water exposures for the planned application of CFT Legumine or Noxfish do not require mitigation measures to protect the public health for any of the receptors evaluated.

Table J-51. Comparison of Piscicide and Neutralization Formulation Component Concentrations in Surface Water to Health-Based Screening Levels and Taste/Odor Threshold Concentrations

Component	Modeled Concentration in Surface Water Following Treatment with Formulation (mg/L)		Health-Based Screening Levels for Surface Water (mg/L)		Taste/Odor Threshold (mg/L)
	CFT Legumine	NoxFish	Child Camper	Unauthorized Youth	
Rotenone	4.21E-02	4.88E-02	3.20E+00	3.70E+00	-
Butylbenzene, 1-	7.80E-05	8.79E-03	1.11E+01	1.25E+01	-
Butylbenzene, sec-	4.00E-06	na	8.54E+00	9.64E+00	-
Isopropylbenzene	na	5.00E-05	1.24E+02	1.41E+02	8.00E-04
Isopropyltoluene, 4-	5.00E-06	9.76E-04	1.09E+02	1.23E+02	-
Methylnaphthalene, 2-	1.36E-04	na	8.80E-01	9.99E-01	2.30E-02
Naphthalene	3.41E-04	6.83E-02	2.46E-01	2.81E-01	2.10E-02
Propylbenzene, 1-	na	3.03E-04	3.00E+01	3.41E+01	-
Toluene	na	1.76E-03	5.40E+02	6.22E+02	4.20E-02
Trichloroethene	na	7.10E-05	3.98E-01	4.70E-01	3.10E-01
Trimethylbenzene, 1,2,4-	na	9.76E-03	1.03E+02	1.17E+02	-
Trimethylbenzene, 1,3,5-	4.00E-06	8.39E-04	7.81E+01	8.84E+01	1.50E-02
Xylene, 1,2-	na	7.40E-05	9.11E+01	1.04E+02	1.70E-02
Xylene, 1,3- and/or 1,4-	na	5.95E-04	8.21E+01	9.38E+01	1.70E-02
Diethylene glycol monoethyl ether	**5.81E-01**	na	3.65E+03	5.25E+03	**2.10E-02**
Methyl-2-pyrrolidinone, 1-	8.78E-02	na	2.68E+02	3.89E+02	-
Rotenolone	5.20E-04	1.46E-02	nd	nd	-
Potassium permanganate	4.00E+00		3.32E+01	4.44E+01	4.44E+01

na = not applicable; nd = not determined.

Conc = modeled surface water concentration exceeds taste/odor threshold.

Conc = modeled surface water concentration exceeds this taste/odor threshold.

J.5.2.3.4 Sediment EPCs Compared to HBSLs

Table J-52 incorporates the sediment maximum concentrations (from Table J-24) to the HBSLs protective of direct sediment contact for all receptors evaluated. Table J-52 shows that the sediment concentrations of the four compounds reported after the previous application of Nusyn Noxfish are all below the HBSLs for both the child camper and the unauthorized youth. Therefore, the potential sediment exposures for the planned application of CFT Legumine does not require mitigation measures to protect the public health for any of the receptors evaluated.

Table J-52. Comparison of Piscicide and Neutralization Formulation Component Concentrations in Sediment to Health-Based Screening Levels

Component	Formulation		Concentration in Sediment After Surface Water Treatment with NuSyn-NoxFish[1] (mg/kg)	Health-Based Screening Levels for Sediment (mg/kg)	
	CFT Legumine	NoxFish		Child Camper	Unauthorized Youth
Rotenone	√	√	2.10E+00	4.8E+02	5.4E+02
Butylbenzene, 1-	√	√	na	1.3E+04	1.5E+04
Butylbenzene, sec-	√		na	1.3E+04	1.5E+04
Isopropylbenzene		√	na	4.8E+04	5.4E+04
Isopropyltoluene, 4-	√	√	na	9.7E+04	1.1E+05
Methylnaphthalene, 2-	√		3.10E-01	3.3E+02	3.6E+02
Naphthalene	√	√	1.46E-01	4.7E+01	5.3E+01
Propylbenzene, 1-		√	na	1.3E+04	1.5E+04
Toluene		√	na	9.7E+04	1.1E+05
Trichloroethene		√	na	3.6E+01	4.1E+01
Trimethylbenzene, 1,2,4-		√	na	6.1E+04	6.8E+04
Trimethylbenzene, 1,3,5-	√	√	na	6.1E+04	6.8E+04
Xylene, 1,2-		√	na	2.4E+04	2.7E+04
Xylene, 1,3- and/or 1,4-		√	na	2.4E+04	2.7E+04
Diethylene glycol monoethyl ether	√		na	7.3E+04	8.1E+04
Methyl-2-pyrrolidinone, 1-	√		na	5.2E+03	5.8E+03
Rotenolone	√	√	3.60E-01	nd	nd
Potassium permanganate			na	7.4E+03	9.4E+03

na = not available.

nd = not determined.

[1] Siepmann and Finlayson, 1999

J.5.3 Risk Uncertainties and Data Gaps

J.5.3.1 Ecological Risks

J.5.3.1.1 Data Gaps, Assumptions and Uncertainties in Environmental Fate and Toxicity Assessment

Ecological toxicity information for some chemicals in the rotenone formulations, and certain organisms was lacking or simply unavailable. In some cases animal toxicity data was available for certain routes (e.g. intravenous) but not for more likely environmental routes such as ingestion and dermal contact, or via inhalation. Even when toxicity information was available for routes of exposure relevant to the Proposed Project and treatment alternatives, often toxicity reference values (TRVs) were unavailable for the specific receptors commonly found around Lake Davis. Therefore, TRVs from typical laboratory species were generally used as surrogates to estimate the potential toxicity to ecological receptors that could be found in the Lake Davis project area. An ideal surrogate species is very similar in both biology and ecology (i.e. in the same guild) to the receptor species and would therefore be a good indicator of response to certain chemicals.

The following bullets highlight the specific data gaps identified in literature review for this assessment, and qualitatively characterize the significance of the uncertainties created by these data gaps

(1) There was essentially no information on rotenone toxicity data found for aquatic or terrestrial plants. Given rotenone is used as an organic pesticide, and is approved for use on over 90 organic food crops (USEPA 2006), with application rates far greater than what could be encountered under the Proposed Project, plant toxicity would appear extremely unlikely.

(2) Chronic rotenone toxicity data on birds was lacking in the literature. As the Proposed Project and treatment alternatives are for a single, short-term treatment, potential chronic exposure is not relevant.

(3) Essentially no information was found on the photo degradation rate of rotenone on soil. This information could be potentially useful to predict exposure to wildlife consuming inadvertently treated vegetation along the waters edge. The uncertainty this data gap creates in estimating dose via this pathway is likely trivial given soil applications will be avoided by the aquatic application of the piscicide.

(4) The toxicity database on reptiles and amphibians remains poor (both for rotenone and most of the formulation constituents). Standard practice is to use avian toxicity data as a surrogate for these species' guilds. Given the respiratory mechanism of action of rotenone, such data are not particularly useful.

(5) Toxicity and empirical fate data on several dispersants in the formulations under consideration was incomplete in the literature. For example, we did not identify inhalation toxicity values for diethylene glycol ethyl ether, absolute degradation rates for permanganate (as a covariate of organic matter), nor dermal toxicity values for most of

the formulation constituents. Although such data would improve the toxicity conclusions in this risk assessment, toxicity comparisons between the technical grade rotenone powder and the formulated end products has shown the latter to be essentially less toxic by at least a factor of 2 (USEPA 2006). Such results indicate that the dispersants in the end-product formulations do not contribute to toxicity (and may actually reduce it).

J.5.3.1.2 Data Gaps, Assumptions and Uncertainties in Ecological Exposure Assessment

For the purposes of estimating exposure point concentrations in water and air, full mixing of all chemicals was assumed. Results from the 1997 Lake Davis treatment (Siepmann & Finlayson 1999) indicate that relatively complete vertical mixing occurred within three days following treatment. This is the most important factor when estimating air concentrations that are generated at the air:water interface. Less important is the horizontal mixing which occurred more slowly and may be an artifact of unequal application of rotenone in the reservoir. Regardless, horizontal mixing occurred within two weeks.

With respect to the calculation of exposure doses from which risks were characterized, several assumptions were implicit that likely overestimate the ingestion doses received by potentially exposed wildlife. These include:

1. The Site Use Factor (SUF) was assumed to be 100% for all receptors. While this assumption may hold true for certain less mobile organisms, this is a very conservative estimate for larger, more mobile receptors.
2. The percent bioavailability of the selected chemical was always assumed to be 100%. Unless a chemical is delivered intravenously to an organism the bioavailability is unlikely to be 100%. Therefore this is a conservative assumption especially as rotenone tends to adhere to sediment and other particles and, as a result, may become unavailable to many organisms. Also the bioavailability of a particular substance can be affected by environmental parameters such as oxygen levels, pH, and temperature.
3. The bioaccumulation factor (BaF) of rotenone in organic tissue was considered to be 1 (i.e., no bioaccumulation) with the exception of fish. Fish were considered to bioaccumulate up to a factor of 20 which reflects the maximum bioaccumulation factor determined by Rach & Gingerich (1986). The volatility and degradability of the inert ingredients comprising the piscicide formulations make them highly unlikely to bioaccumulate in organic matter and were therefore assumed to have a BaF of 1.
4. The percent contaminated food was always assumed to be 100% indicating that all food sources were contaminated. This is a conservative assumption as most organisms would have alternate foraging opportunities.
5. Where species-specific data relating to food and water intake was not available, intake rates of food, water and air as well as surface area were estimated for each ecological receptor using allometric formulae taken from the Wildlife Exposure Handbook (USEPA 1993) and Sample et al. (1996). These formulae use a species' average weight to determine said rates. These numbers can exhibit a great deal variation among populations, but population-specific data from the Lake Davis area were not available.

J.5.3.2 Human Receptor Exposure and Risk Characterization

The procedures used in any quantitative risk assessment result in estimates of HBSL values that are based on many conservative assumptions about exposure and toxicity. Using site-specific factors as was done for this project decreases uncertainty, although uncertainty persists in even the most site-specific risk assessments. The inherent uncertainty in quantitative risk assessment methodology affects the level of confidence that can be placed in the final results. The inherent uncertainty and reasonableness of the assumptions must be considered concurrently with the estimated risk values when using the findings of a risk assessment for risk management decisions.

Using assumptions that tend to overestimate exposure and, therefore, lower HBSL values, increases the degree of certainty in the health protection provided by risk mitigation measures based on those HBSLs. The RME assumptions used in developing the HBSLs are intended to provide an upper bound exposure evaluation, to provide a high degree of certainty for the health protection offered by those values.

The risk measurements used in EIRs, are not full probability estimates of risk but are conditional estimates given a set of assumptions about exposure and toxicity. Therefore, it is important to specify the assumptions and uncertainties inherent in the HBSLs to place the risk estimates in proper perspective. A qualitative uncertainty analysis of each risk assessment component is sufficient for most projects. Specific sources of uncertainty in this risk assessment are discussed in the following sections.

J.5.3.2.1 Uncertainties Associated with Data Evaluation

The data used for this risk assessment consisted of piscicide formulation information proposed for this project, thus, there is a high level of confidence in understanding the components of the formulations to be included in the risk assessment to include the active ingredients, adjuvants, carriers and inert ingredients. Historical information on previous fish eradication projects conducted at the site, provided insight on what key aspects of the formulations required more detailed evaluation in the risk assessment. For example, historically there have been concerns on harmful odors generated from project as well as concerns that the piscicide formulations may pose unacceptable health risks to human populations recreating or working near or at the lake. As a result, the risk assessment focused on all completed potential exposure pathway to ensure that the public concerns were addressed and that the risk evaluation was conducted in the most comprehensive manner possible. Finally, using reasonable maximum conditions for all constituents identified in the formulation which have toxicity information available, for risk estimation purposes, the risks are expected to more likely be overestimated than underestimated.

J.5.3.2.2 Uncertainties Associated with the Exposure Assessment

The HBSLs were derived from estimates for the RME scenarios. These chemical intakes are conditional estimates that include numerous assumptions on the type of exposures that may occur, the frequency and duration of those exposures, and the concentration of piscicide constituents at the point of exposure. This standard approach is intended to provide a

conservative estimate of intake, more likely to overestimate than underestimate site-related risk. Relatively conservative assumptions are used for many of the exposure parameters, resulting in a compounding effect.

One major area of uncertainty in the exposure assessment is the prediction of human activities that may lead to contact with constituents in environmental media. The degree to which this exposure assessment fully reflects the activities and processes that may lead to contact with piscicide constituents in environmental media cannot be estimated. Activities that differ from the assumptions made for a particular pathway could lead to exposures different than those quantified. However, the exposure assumptions for this project were based predominately on RME or upperbound values from guidance where site-specific information was not available. This approach was used in order to develop HBSLs that are conservative and health protective. The probability of occurrence was not included in the quantification of risk. If an exposure scenario does not occur, the risk as calculated will not occur.

Exposure point concentrations are inherently uncertain because piscicide constituent concentrations are assumed to remain constant over the period of exposure when all the piscicide constituents are known to have short half-lives in water and air. This assumption likely overestimates potential exposure to organic constituents that will decrease over time due to degradation processes. The assumptions that the measured concentrations are constant over the duration of exposure may overestimate the intake and associated risk.

J.5.3.2.3 Uncertainties Associated with the Human Toxicity Assessment

While the method used for the development of CSFs assumes a nonthreshold approach, experimental evidence indicates that some of the potential carcinogens have dose-response curves that suggest a response threshold. The assumption of a non-threshold response can lead to conservative errors, where risk is overestimated.

Another source of uncertainty in a risk assessment is associated with the use of dose-response data generated under experimental laboratory conditions (using non-human mammals) and extrapolating these results for comparison to human exposure under a different environmental exposure scenario. To extrapolate the experimental evidence from animals to humans, a series of uncertainty factors and modifying factors, which have been derived by USEPA, are applied. These uncertainty factors and modifying factors are the quantitative uncertainty associated with the value for each specific chemical. The greater the uncertainty factor/modifying factor, the greater the uncertainty behind applicability of the value to humans under environmental exposure conditions. Uncertainty factors are typically assigned by USEPA in a conservative manner, so that the toxicity values tend to overestimate the potential for adverse effects.

For ingestion exposures, the bioavailability of constituents in the human body is assumed to be the same as that in the study organisms from which toxicity factors were developed. Most toxicity parameter values are calculated to be used with administered rather than absorbed doses; however, their values still reflect the bioavailability of the as-administered form.

HBSLs are likely to be lower than needed if constituent bioavailability from environmental media is less than that from the experimentally administered doses in toxicological studies.

Dermal toxicity values are not available for use in estimating risk from direct contact. HBSLs must be estimated using oral toxicity values adjusted from administered to absorbed dose; however, this does not account for response differences between oral and dermal doses. Dermal doses are not subject to first-pass hepatic metabolism before reaching the systemic circulation. The resulting dermal dose may be greater than a dose received orally; or, if the toxic effect is attributable to an active metabolite, may be more pronounced than if received orally. The uncertainty involved in using an oral slope factors for dermal absorption may contribute to either overestimation or underestimation of risk depending on the chemical and how it is metabolized.

J.5.3.2.4 Uncertainties Associated with the Human Health Risk Characterization

The uncertainties associated with estimating cancer risk and noncancer hazard are primarily those built into the process of deriving the estimates, as previously discussed. Multiple pathway exposures were incorporated into the HBSLs along with conservative toxicity values selected to be health protective. Therefore, the HBSLs developed for this project are likely to be lower than those concentrations that would be health protective for each piscicide constituent evaluated. Possible interactions (antagonistic or synergistic) that could occur among the various piscicide constituents present are not included in this evaluation. Because the proposed formulations used are well-studied, it is not anticipated that interactions could result in underestimating the health protective concentration for the project.

In most cases, the uncertainties in the risk assessment are compensated for by inclusion of upperbound exposure factors, uncertainty factors, and modifying factors in developing RfDs and CSFs, and by assuming that degradation of the piscicide constituents does not occur during the project. Incorporation of factors and variables to account for uncertainty in each step of the risk assessment process results in conservative and health protective HBSLs. This procedure ensures the protection of public health, because if the project concentrations of piscicide constituents do not exceed the HBSL, then there is a high level of confidence that an adverse impact will not occur.

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